A COMPARATIVE ASSESSMENT OF SONOSALPINGOGRAPHY (SSG) AND DIAGNOSTIC LAPAROSCOPY FOR DETERMINATION OF TUBAL PATENCY IN CASES OF PRIMARY AND SECONDARY SUBFERTILITY

Dissertation submitted to

THE TAMILNADU

Dr.M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the requirement
for the award of

M.S.DEGREE – OBSTETRICS & GYNAECOLOGY

BRANCH - II

GOVERNMENT KILPAUK MEDICAL COLLEGE
KILPAUK, CHENNAI.

APRIL 2015

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"A COMPARATIVE ASSESSMENT OF SONOSALPINGOGRAPHY (SSG) AND DIAGNOSTIC LAPAROSCOPY FOR DETERMINATION OF TUBAL PATENCY IN CASES OF PRIMARY AND SECONDARY SUBFERTILITY"** is the bonafide original work of **Dr.RAJANI.C** under the guidance of **Dr.P.S.JIKKI KALAISELVI, MD.,DGO.,** Associate Professor of department of Obstetrics and Gynaecology KMCH, Chennai in partial fulfillment of the requirements for MS Obstetrics and Gynaecology branch II examination of the Tamilnadu Dr.MGR Medical university to be held in April 2015 .The period of Postgraduate study and training from June 2012 to April 2015.

Prof. DR.P.S.JIKKI KALAISELVI MD.,DGO,
Associate Professor,
Department of Obstetrics and Gynaecology,
Kilpauk Medical College.

Prof.Dr.T.K SHAANTHY GUNASINGH MD.,DGO,
Professor and Head of the Department,
Department of Obstetrics and Gynaecology,
Kilpauk Medical College.

Prof.Dr.N.GUNASEKARAN,M.D.,D.T.C.D.,
The Dean
Government Kilpauk Medical College,
Chennai – 600 010.

DECLARATION

I solemnly declare that this dissertation **"A COMPARATIVE ASSESSMENT OF SONOSALPINGOGRAPHY (SSG) AND DIAGNOSTIC LAPAROSCOPY FOR DETERMINATION OF TUBAL PATENCY IN CASES OF PRIMARY AND SECONDARY SUBFERTILITY"** was prepared by me at Government Kilpauk Medical College and hospital,Chennai, under the guidance of **Dr.P.S.Jikki Kalaiselvi MD.,DGO,** Professor ,Department of Obstetrics and Gynaecology,Government Kilpauk Medical College and hospital, Chennai.

This dissertation is submitted to **The Tamil Nadu Dr.M.G.R. Medical University, Chennai** in partial fulfillment of the University regulations for the award of the degree of **M.S Obstetrics and Gynaecology.**

Place:

Date: **(Dr.C.Rajani)**

ACKNOWLEDGEMENT

I start this thesis in the name of Almighty God, the most beneficent and forgiving. I thank God that he has given me the privilege to learn from the able teachers in my department.

I express my sincere thanks to **Dr.N.GUNASEKARAN M.D.,D.T.C.D.,** Dean, Kilpauk Medical College for allowing me to conduct the study using the available facilities.

I convey my heartfelt gratitude and sincere thanks to my guide **Dr. P.S. JIKKI KALAISELVI**, **M.D.**, **D.G.O.**, Professor, Department of Obstetrics and Gynaecology, Kilpauk Medical College who with her exhaustive knowledge and Professional expertise has provided able guidance and constant encouragement throughout the Course of my study and in the preparation of this dissertation.

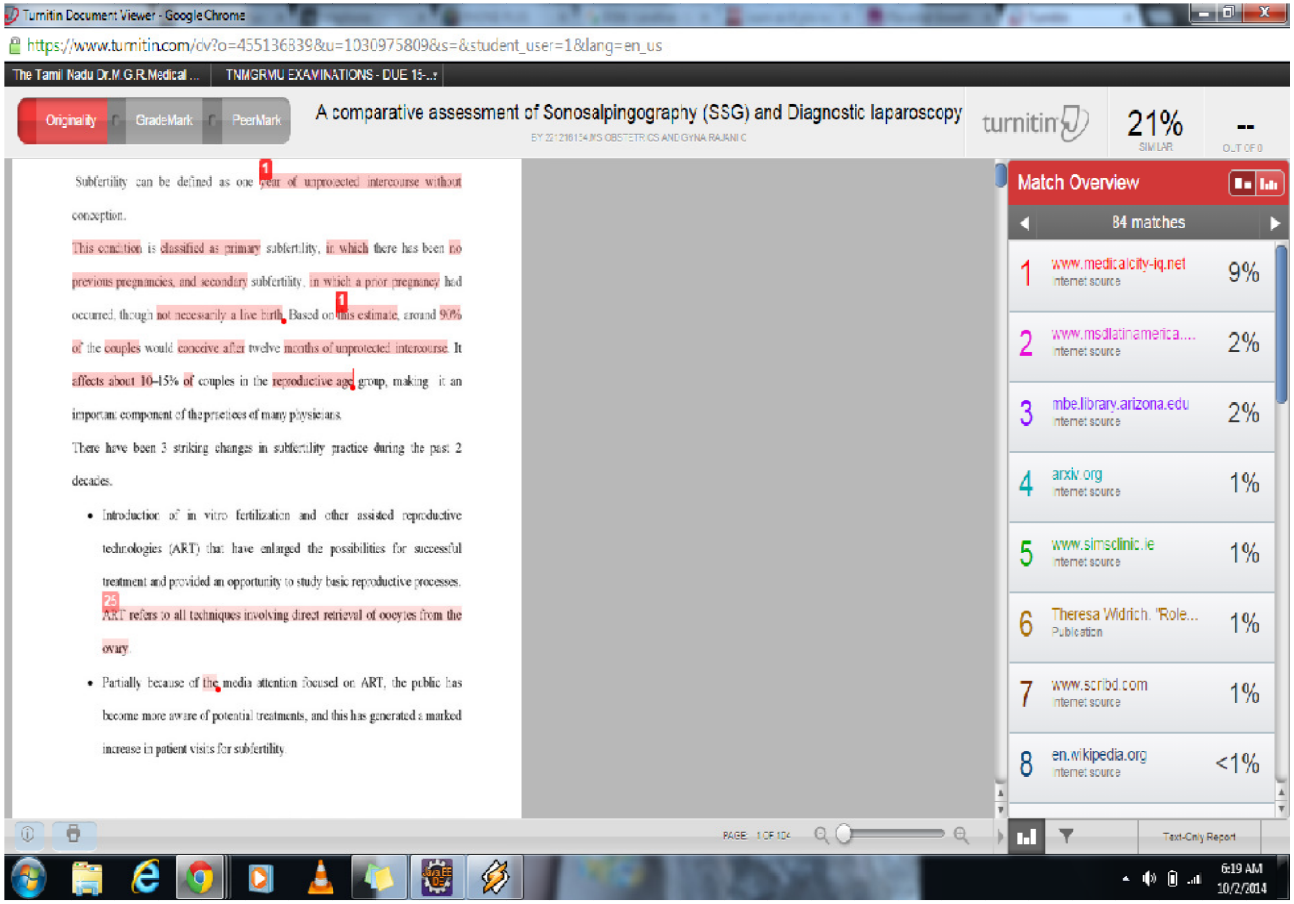
I express my sincere thanks to my professor **Dr.T.K.SHAANTHY GUNASINGH**, **M.D.**, **D.G.O.**, Professor and Head of the department of Obstetrics and Gynaecology and **Dr.SRIMATHY**, **M.D.**, **D.G.O.**, Registrar, Department of Obstetrics and Gynaecology, Kilpauk Medical College, Kilpauk, Chennai, for their valuable help and encouragement.

I am grateful to my Assistant Professors, colleagues and my friends for their advice and suggestions.

My special thanks to my husband **Mr.A.RAJESH** for supporting me through my career in every possible way.

My heartful thanks to my parents, my family and friends, who have been a constant source of encouragement and immense help, for instilling in me a sense of commitment and for their belief in me.

Last but not least I thank all **my PATIENTS**, who formed the backbone of this study without them this study would not have been possible.

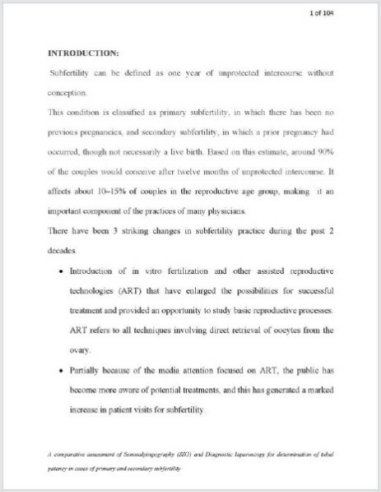

Turnitin Document Viewer - Google Chrome
https://www.turnitin.com/dv?o=455136839&u=1030975809&s=&student_user=1&lang=en_us
The Tamil Nadu Dr.M.G.R.Medical ...
TNMGRMU EXAMINATIONS - DUE 15-...
Originality
GradeMark
PeerMark
A comparative assessment of Sonosalpingography (SSG) and Diagnostic laparoscopy
BY 221216154 JMS OBSTETRICS AND GYNA RAJANI C
turnitin
21%
SIMILAR
--
OUT OF 0
Subfertility can be defined as one year of unprotected intercourse without conception.
This condition is classified as primary subfertility, in which there has been no previous pregnancies, and secondary subfertility, in which a prior pregnancy had occurred, though not necessarily a live birth. Based on this estimate, around 90% of the couples would conceive after twelve months of unprotected intercourse. It affects about 10-15% of couples in the reproductive age group, making it an important component of the practices of many physicians.
There have been 3 striking changes in subfertility practice during the past 2 decades.
Introduction of in vitro fertilization and other assisted reproductive technologies (ART) that have enlarged the possibilities for successful treatment and provided an opportunity to study basic reproductive processes.
ART refers to all techniques involving direct retrieval of oocytes from the ovary.
Partially because of the media attention focused on ART, the public has become more aware of potential treatments, and this has generated a marked increase in patient visits for subfertility.
Match Overview
84 matches
1 www.medicalcity-iq.net Internet source 9%
2 www.msdlatinamerica.... Internet source 2%
3 mbe.library.arizona.edu Internet source 2%
4 arxiv.org Internet source 1%
5 www.simsclinic.ie Internet source 1%
6 Theresa Widrich, "Role... Publication 1%
7 www.scribd.com Internet source 1%
8 en.wikipedia.org Internet source <1%
PAGE 1 OF 104
Text-Only Report
6:19 AM
10/2/2014

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	221216154.ms Obstetrics And Gyna...
Assignment title:	TNMGRMU EXAMINATIONS
Submission title:	A comparative assessment of Sonos...
File name:	Final_Dissertation_for_pliagirism.docx
File size:	4.31M
Page count:	104
Word count:	11,264
Character count:	68,065
Submission date:	01-Oct-2014 01:20AM
Submission ID:	455136839



1 of 104

INTRODUCTION:

Subfertility can be defined as one year of unprotected intercourse without conception.

This condition is classified as primary subfertility, in which there has been no previous pregnancies, and secondary subfertility, in which a prior pregnancy had occurred, though not necessarily a live birth. Based on this estimate, around 90% of the couples would conceive after twelve months of unprotected intercourse. It affects about 10-15% of couples in the reproductive age group, making it an important component of the practices of many physicians.

There have been 3 striking changes in subfertility practice during the past 2 decades.

- Introduction of in vitro fertilization and other assisted reproductive technologies (ART) that have enlarged the possibilities for successful treatment and provided an opportunity to study basic reproductive processes. ART refers to all techniques involving direct retrieval of oocytes from the ovary.
- Partially because of the media attention focused on ART, the public has become more aware of potential treatments, and this has generated a marked increase in patient visits for subfertility.

A comparative assessment of Sonosalpingography (SSG) and Diagnostic laparoscopy for determination of tubal patency in cases of primary and secondary subfertility

CONTENTS

INTRODUCTION

Subfertility can be defined as one year of unprotected intercourse without conception.

This condition is classified as primary subfertility, in which there has been no previous pregnancies, and secondary subfertility, in which a prior pregnancy had occurred, though not necessarily a live birth. Based on this estimate, around 90% of the couples would conceive after twelve months of unprotected intercourse. It affects about 10–15% of couples in the reproductive age group, making it an important component of the practices of many physicians.

There have been 3 striking changes in subfertility practice during the past 2 decades.

- Introduction of in vitro fertilization and other assisted reproductive technologies (ART) that have enlarged the possibilities for successful treatment and provided an opportunity to study basic reproductive processes. ART refers to all techniques involving direct retrieval of oocytes from the ovary.

- Partially because of the media attention focused on ART, the public has become more aware of potential treatments, and this has generated a marked increase in patient visits for subfertility.
- Increase in the proportion of women over 35 seeking medical attention for subfertility.

Some of the main causes of subfertility are male factors, ovulatory disorders ovulatory factors, diminshed ovarian reserve, tubal injury or blockage ,or paratubal adhesions (which includes endometriosis with evidence of tubal or peritoneal adhesions), uterine causes, systemic conditions like infections or chronic diseases , autoimmune conditions and chronic renal failure, cervical and immunologic factors, and unexplained factors.Few basic investigations that are to be performed before starting any subfertility treatment are confirmation of ovulation, semen analysis, and the documentation of tubal patency. Since either or both partners may contribute subfertility ; it is important to rule out all diagnosis before pursuing invasive treatment.

REVIEW OF LITERATURE

Table 30.1 Causes of Infertility	
Relative prevalence of the etiologies of infertility (%)	
Male factor	25–40
Both male and female factors	10
Female factor	40–55
Unexplained infertility	10
Approximate prevalence of the causes of infertility in the female (%)	
Ovulatory dysfunction	30–40
Tubal or periotoneal factor	30–40
Unexplained infertility	10–15
Miscellaneous causes	10–15

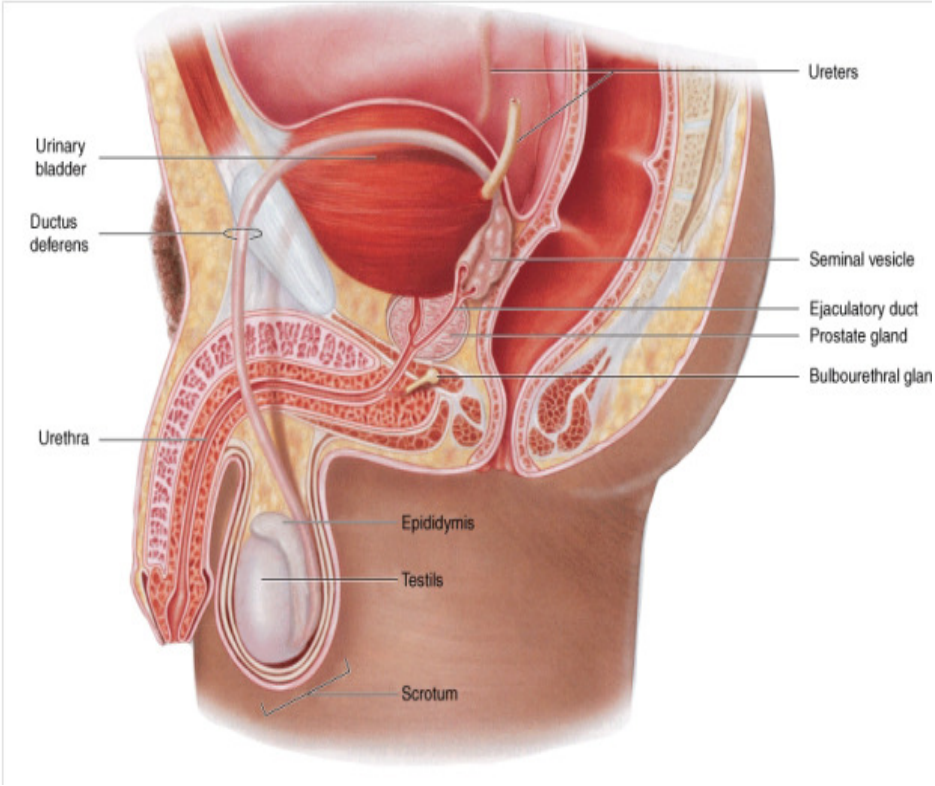
Male Factor:

Male factors contribute to subfertility in 20% of subfertile couples, but it could be the cause in as many as 30% to 40% of cases. The interpretation and value of the semen analysis and other tests for male subfertility is mandatory in the evaluation of male factor of subfertility.

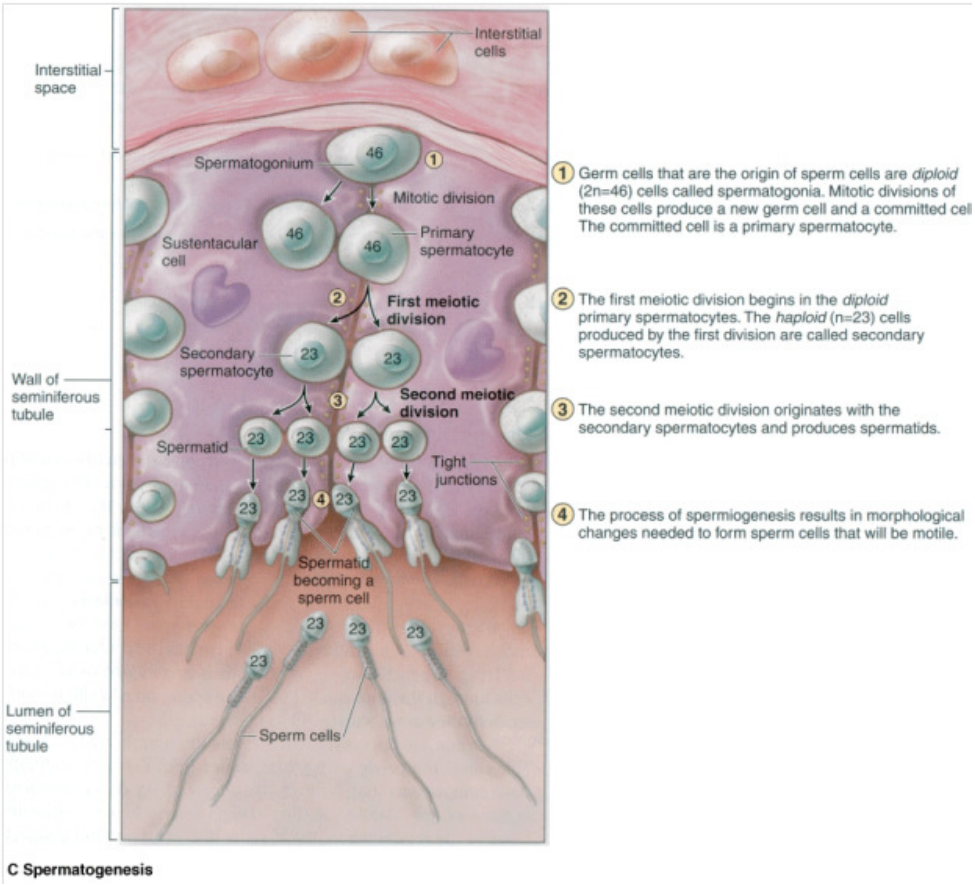
Physiology:

The male reproductive tract consists of the testis, the vas deferens, epididymis, prostate, the ejaculatory ducts, seminal vesicles, bulbourethral glands, and the urethra. Testes consist of two types of cell:

- Sertoli cells, lining the seminiferous tubules which is the the site of spermatogenesis
- and Leydig cells which is the site of androgen synthesis.



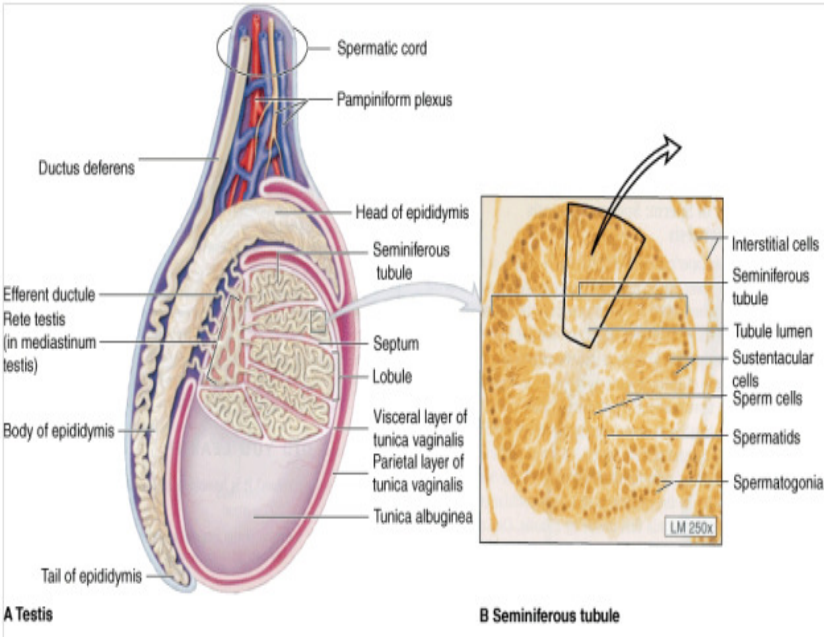
In man, the two hormones namely, follicle–stimulating hormone and luteinizing hormone are secreted by the pituitary gland. The luteinizing hormone helps in the synthesis and secretion of testosterone by the Leydig cells, while the follicle–stimulating hormone causes the Sertoli cells to secrete inhibin. Both testosterone and follicle stimulating hormone act on the seminiferous tubules to help in spermatogenesis. The period for development of spermatogonia from mature stem cells is around 75 days. Spermatogonia undergo mitotic division to produce spermatocytes. The spermatocytes then further undergo meiotic division to produce spermatids, containing haploid number of chromosomes.



C Spermatogenesis

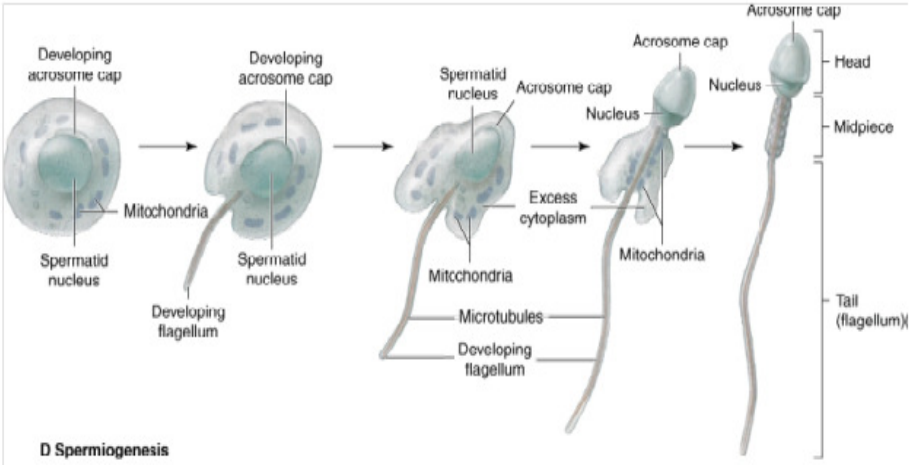
After attaining maturity, these spermatids become spermatozoa and enter the epididymis, where they continue to mature. These spermatids become progressively more motile during the 12 to 21 days they travel through the epididymis.

Following ejaculation, mature spermatozoa along with fluid from the prostate, seminal vesicles, and bulbourethral glands, get released from the vasdeferens.



The semen is a combination of spermatozoa and the seminal plasma. Following ejaculation, it thins out 20 to 30 minutes by liquefaction process due to the action of proteolytic enzymes in the prostatic fluid. For fertilization to occur, “capacitation”, should take place within the sperm's outer surface membrane. This process usually occurs in the cervical mucus but it can also occur in physiologic media in vitro.

Finally, at the time of fertilization, the sperm undergoes the " acrosome reaction", which involves the release of enzymes of the inner acrosomal membrane resulting in the breakdown of the outer plasma membrane and thus, the fusion with outer acrosomal membrane. Subsequent fusion between the ovum and sperm after the penetration of the ovum's zona pellucida is brought about by the "acrosome reaction" and binding of sperm and ovum surface proteins. As the sperm penetrates the egg, "cortical reaction" initiates a hardening of the zona pellucida thus preventing the penetration of additional sperms.



Cocaine and Marijuana use reduces sperm concentration. Certain drugs have been seen to reduce function, sperm count or cause ejaculatory dysfunction. Use of caffeine, smoking and alcohol have been associated with reduced semen quality or quantity in a dose–related fashion.

Drugs that Can Impair Male Fertility

1	Impaired spermatogenesis	sulfasalazine, nitrofurantoin, colchicine, methotrexate, chemotherapy
2	Pituitary suppression	gonadotrophin–releasing hormone analogues, testosterone injections
3	Antiandrogenic effects	spironolactone ,cimetidine
4	Ejaculation failure	antidepressants ,α blocker , phenothiazines
5	Erectile dysfunction	thiazide diuretics, β blockers, metoclopramide
6	Drugs of misuse	cannabis, heroin, β anabolic steroids, cocaine

Semen Analysis

The basic semen analysis includes volume of semen, concentration of sperms, its motility and morphology,viscosity, fructose levels, and WBC counts within the semen.

Specimen Collection

The specimen obtained by masturbation after 2-3 days of abstinence is collected in a clean container. The specimens are maintained at body temperature and are taken to the laboratory within half to one hour of collection.

Semen Analysis Terminology and Normal Values

Semen analysis terminology

1	**Normozoospermia**	All the parameters normal
2	**Oligozoospermia**	Reduction in the sperm numbers Mild to moderate: 5–20 million/mL of semen Severe: <5 million/mL of semen
3	**Teratozoospermia**	Increase in the abnormal forms of sperm
4	**Oligoasthenoteratozoospermia**	Presence of reduced number and abnormal forms of sperms
5	**Azoospermia**	Absence of sperms in semen
6	**Aspermia (anejaculation)**	No semen (ejaculation failure)
7	**Leucocytospermia**	Increased WBCs in semen
8	**Necrozoospermia**	non–viable sperms
9	**Asthenozoospermia**	Reduction in the sperm motility

Normal seminal fluid analysis (World Health Organization)

1	Volume	More than 2 mL
2	Sperm concentration	More than 20 million/mL
3	Sperm motility	More than 50% progressive or More than 25% rapidly progressive
4	Morphology (strict criteria)	More than 15% normal forms
5	White blood cells	More than 1 million/mL

Table 30.4 Etiologic Factors in Male Infertility

Pretesticular	*Testicular*
Endocrine	***Genetic***
Hypogonadotropic hypogonadism	Klinefelter's syndrome
Coital disorders	Y chromosome deletions
Erectile dysfunction	Immotile cilia syndrome
Psychosexual	***Congenital***
Endocrine, neural, or vascular	Cryptorchidism
Ejaculatory failure	***Infective (orchitis)***
Psychosexual	***Antispermatogenic agents***
After genitourinary surgery	Heat
Neural	Chemotherapy
Drug related	Drugs
Posttesticular	Irradiation
Obstructive	***Vascular***
Epididymal	Torsion
Congenital	Varicocele
Infective	***Immunologic***

Vasal	*Idiopathic*
Genetic: cystic fibrosis	
Acquired: vasectomy	
Epididymal hostility	
Epididymal asthenozoospermia	
Accessory gland infection	
Immunologic	
Idiopathic	

FEMALE FACTORS:

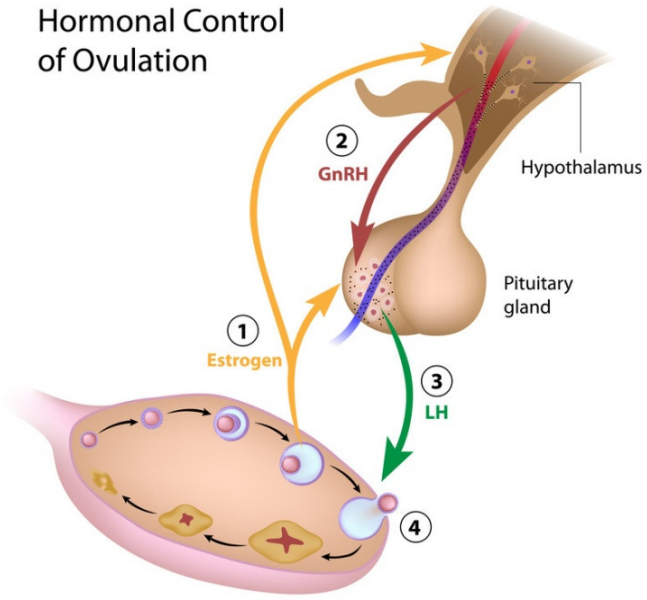
Chronologic age is one of the most important predictors of ovarian reserve and so remains a strong factor in the success of both spontaneous and ART cycles. Ovarian reserve gives an estimate of the number of the actual growing follicles and their reproductive potential. It affects the response of ovaries to exogenous gonadotropins by the measurement of serum estradiol levels,follicle count, duration of stimulation, oocytes produced, and the quantity of exogenous gonadotropins. Screening tests used in the prediction of ovarian responsiveness to controlled ovarian hyperstimulation include

- Serum day 3 Follicle Stimulating Hormone, serum mullerian–inhibiting substance (MIS), serum inhibin B
- Mean ovarian volume measurement and antral follicle count.

- Day 3 follicle stimulating hormone measurement which is based on the fact that a small increase in the basal serum follicle stimulating hormone levels is correlated with the decreased fertlity seen in women in their late 30s.
- Clomiphene citrate challenge test
- Following pituitary desensitization, for IVF treatment basal Follicle stimulating hormone assessment is a better predictor of ovarian response. Clomiphene citrate is known to have some antiestrogenic effects on the hypothalamic–pituitary axis hence the suppression of follicle stimulating hormone production by the pituitary is decreased. The clomiphene citrate challenge test involves the measurement of estradiol and serum follicle stimulating hormone on day 3 and day 10 after administration of clomiphene citrate (100 mg orally each day) from days 5 to 9. The incidence of an abnormal clomiphene citrate challenge test is less than 10% in less than 35 years of age to 26% in more than 40 years of age. Both clomiphene citrate challenge test and day 3 follicle stimulating hormone results display high specificity (96%) in the prediction of IVF outcome. Starting at the preantral follicle stage, Serum inhibin B is secreted by ovarian granulosa cells.During the luteal–follicular transition Inhibin B secretion increases.It reflects

the overall granulosa cell function of the controlled ovarian hyperstimulation of follicles that is recruited to undergo gonadotropin – dependent growth. The production of follicle stimulating hormone by the pituitary gland is suppressed by Inhibin B. In clomiphene citrate challenge test, the main mechanism for suppression of follicle stimulating hormone is inhibin B production by granulosa cells. Of late, MIS, also known as antimullerian hormone (AMH), is investigated for ovarian responsiveness to stimulation and as a marker for ovarian reserve. It is produced by the granulosa cells of follicles both preantral and small antral ,which inhibits the initiation of primordial follicle growth. The level of MIS in women with regular and normal cycles reduces with age and becomes undetectable at the time of menopause. The serum MIS concentration also decreases as the ovarian primordial follicle count decreases, thus making this hormone an early detector of ovarian reserve depletion. Poor ovarian response in ART cycles is associated with decreased levels of MIS .

Ovulatory Factor:

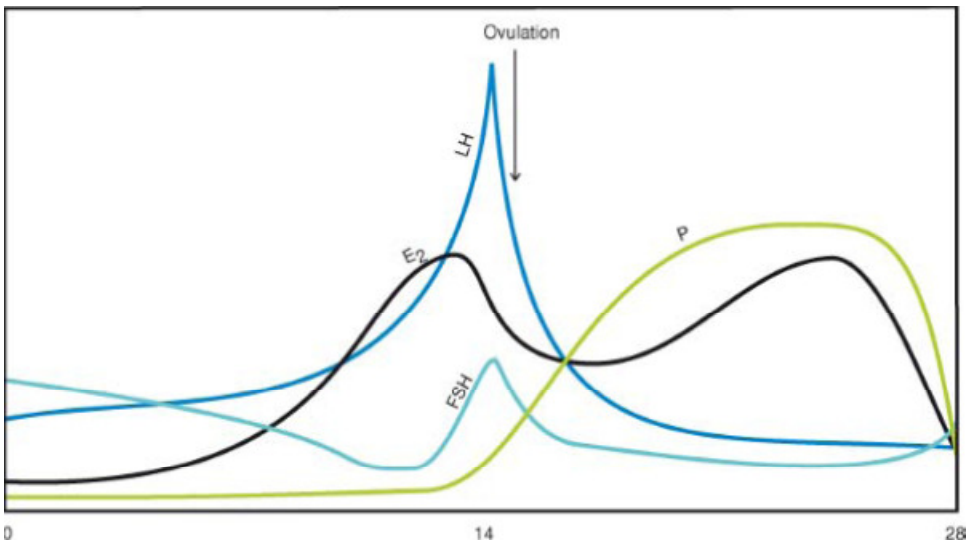


Ovarian disorders cause 30% to 40% of all cases of female subfertility. The normal length of the menstrual cycle in reproductive age women is between twenty five to thirty five days. As ovulation is a prerequisite to conception, ovulation is done as part of the basic assessment of the subfertile couple. Initial diagnoses among women with ovulatory factor subfertility may include oligoovulation (infrequent ovulation) or anovulation (complete absence of ovulation) .

Methods to Document Ovulation

Luteinizing Hormone Monitoring

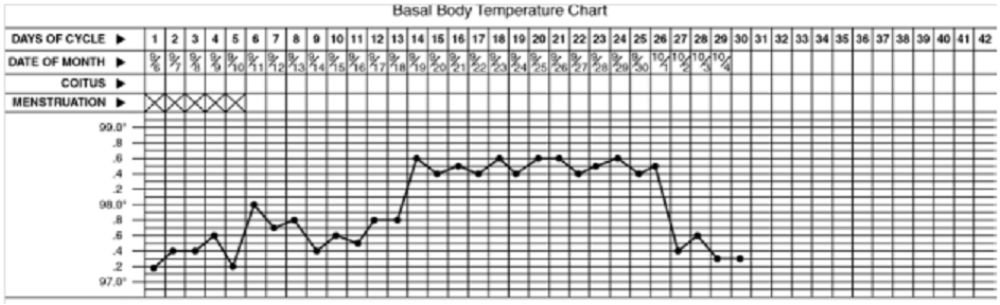
About 34-36 hours following an luteinising hormone surge and around ten to twelve hours following luteinising hormone peak, ovulation occurs.



Relative hormonal fluctuations in a normal, ovulatory, 28–day menstrual cycle.

Basal Body Temperature

Progesterone increases the body temperature by about 0.5°F above the baseline temperature .It is recorded during the follicular phase of the menstrual cycle.

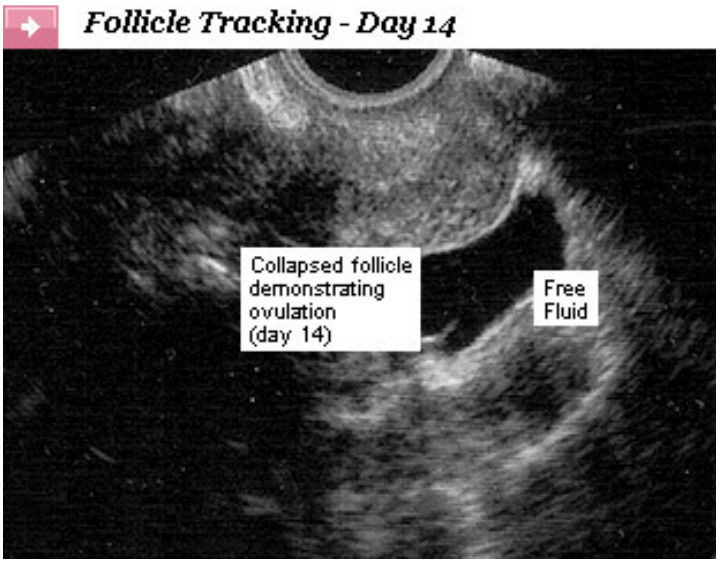


Midluteal Serum Progesterone

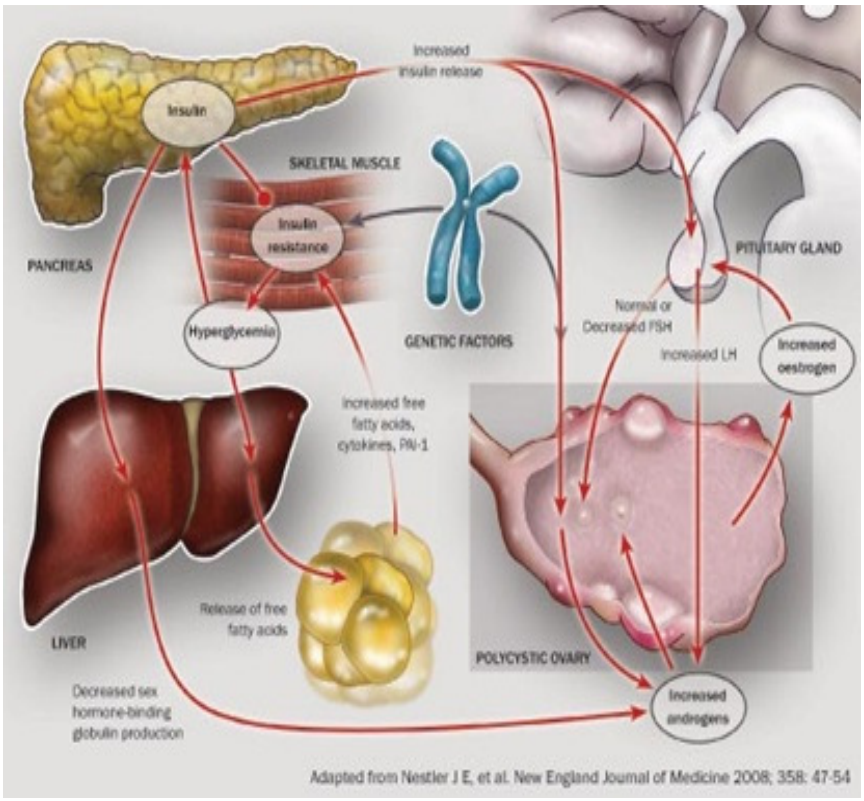
Serum progesterone levels above 3 ng/mL confirm ovulation in the midluteal phase.

Ultrasound Monitoring

Ovulation is characterized by the presence of fluid in the cul–de–sac and a progressive reduction in the size of an ovarian follicle.



Polycystic Ovarian Syndrome



PCOS is the most common cause of anovulation and oligo ovulation— among women presenting with subfertility and in the general population. The presence of two of the following conditions helps in diagnosing PCOS :

●anovulation or oligoovulation

●Hyperandrogenemia

●Hyperandrogenism (clinical manifestations)

●USG findings

As oligomenorrheic women are at high risk for endometrial hyperplasia, it is advocated to do an endometrial biopsy before ovulation induction therapy is begun.

Symptoms of PCOS
Infertility - difficulty getting pregnant
Infrequent / absent / irregular periods
Hirsuitism - increased hair growth
Acne, or oily skin
Male pattern baldness or thinning of the hair
Obesity, weight gain, especially around the waist
More than 12 cysts in one ovary on ultrasound appearance
Skin tags - excess tags of skin under the arms or around the neck
Acanthosis Nigrans - dark pigmented patches on the skin in areas of the nape of the neck, inner thighs, and arms

Table 30.7 Clinical Findings that Suggest Insulin Resistance and Hyperinsulinemia

Physical findings associated with insulin resistance
Body mass index >27 kg/m^2
Waist-to-hip ratio >0.85
Waist >100 cm
Acanthosis nigricans
Numerous achrochordons (skin tags)

From **Barbieri RL.** Induction of ovulation in infertile women with hyperandrogenism and insulin resistance. *Am J Obstet Gynecol* 2000;183:1412–1418, with permission.

Hyperprolactinemia

It is another factor that is associated with ovulatory causes. After excluding pituitary macroadenoma and other intracranial pathologies, correction of the hyperprolactinemic state with *bromocriptine* restores ovulation in about 90% of patients . Cabergoline can also be given as an alternative.

Hypogonadotropic Hypogonadism

It is defined by anovulation in the presence of low serum luteinising hormone, follicle stimulating hormone, and estradiol levels. It reflects dysfunction within the hypothalamic–pituitary axis. Other factors like craniopharyngiomas, pituitary adenomas, AV malformations and other central space–occupying lesions, is excluded using MRI. By disrupting hypothalamic function, low BMI is thought to cause anovulation and subfertility in women. Anorexia nervosa, malnutrition and rigorous athletic training are some examples. Other hypothalamic causes like *Kallmann syndrome* (congenital hypothalamic failure), is treated using pulsatile GnRH therapy,provided the pituitary–ovarian axis is intact.

Hypothyroidism

Abnormal TSH levels among subfertility population was reported to be 1.5%,2.6%,4.8% and 6.3% for women diagnosed with male subfertility, tubal subfertility, unexplained subfertility, and anovulatory subfertility respectively . However when euthyroid status was achieved using *thyroxine* supplementation, they resumed spontaneous ovulation.

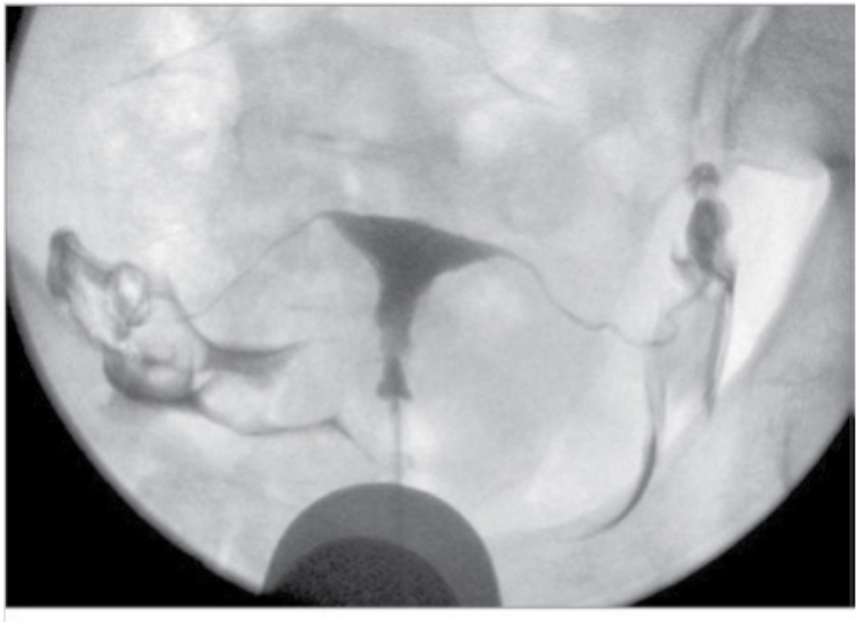
Tubal, Paratubal, and Peritoneal Factors

Peritoneal and Tubal factors cause 30% to 40% of cases of female subfertility. The incidence of tubal subfertility is found to be 54%, 23%, and 12% after three, two, and one episode of PID, respectively. Inspite of it,nearly half of the patients with tubal damage have no identifiable risk factors for tubal disease.

HYSTEROSALPINGOGRAPHY:

It is used as the initial diagnostic test used to assess tubal patency. Other causes of apparent tubal blockage include benign polyps within the tubal lumen, salpingitis ,isthmica nodosa, , intratubal mucous debris tubal endometriosis, tubal spasm. It is generally performed between menstrual days 6 and 11.

Normal HSG:



There is a risk of 0.3% to 1.3% of infectious sequelae after HSG. However in high risk populations it can be as high as 3% .Therefore, PID, cervicitis, known hydrosalpinx and palpable adnexal tenderness or masses on bimanual examination are all contraindications to HSG. Other complications of HSG include hemorrhage, uterine perforation, cervical laceration, allergic response to the contrast dye and vasovagal reaction.

Other Diagnostic Modalities

Falloposcopy allows direct fiberoptic visualization of intratubal architecture, tubal ostia , even intraluminal debris ,abnormal tubal mucosal patterns and tubal ostial spasm which cause tubal obstruction.

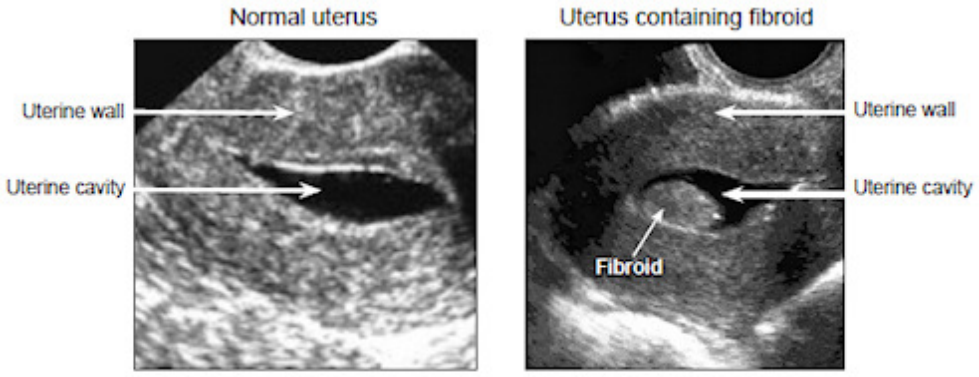
SALINE INFUSION SONOSALPINGOGRAPHY (SIS):

Parsons and Lense in 1993 were the first to develop the technique of using saline as a contrast agent to view the lesions inside the uterine cavity. It has now widely used and is referred to as sonosalpingography (SSG) or saline infusion sonohysterography (SIS). It is a very valuable technique used for the detection of any structural abnormalities such as polyps, submucous myomas and adhesions. Cervix is cleansed with antiseptic solution and a small catheter is placed in the cervical os using a sterile uterine packing forceps till the fundus is reached.Saline is slowly infused into the uterus while observing for uterine distension. The relation of a submucous fibroid to the uterine wall can be defined after the uterine distension. Polyps are seen as echogenic masses in the uterine cavity when outlined by intracavitary fluid that originate from the endometrium with a small or broad base. When the uterine cavity is distended ,adhesions or a septum are easily seen and are outlined by the contrast agent. SSG causes minimal discomfort and a low rate of complications compared to other techniques. Two recent studies evaluated SSG for infertility patients.

Soares et al: “he evaluated the diagnostic accuracy of SIS in uterine cavity pathology in 65 subfertile patients,thus comparing its results with that of hysterosalpingography (HSG) and transvaginal USG.

Hysteroscopy was the gold standard and SIS had the same diagnostic accuracy for endometrial hyperplasia and polypoid lessons”

Gronlund et al: “Using hysteroscopy as the standard ,he assessed the diagnostic value of SIS in the evaluation of metrorrhagia and infertility.SIS had the same sensitivity and specificity with lesser side effects as that of hysteroscopy”.



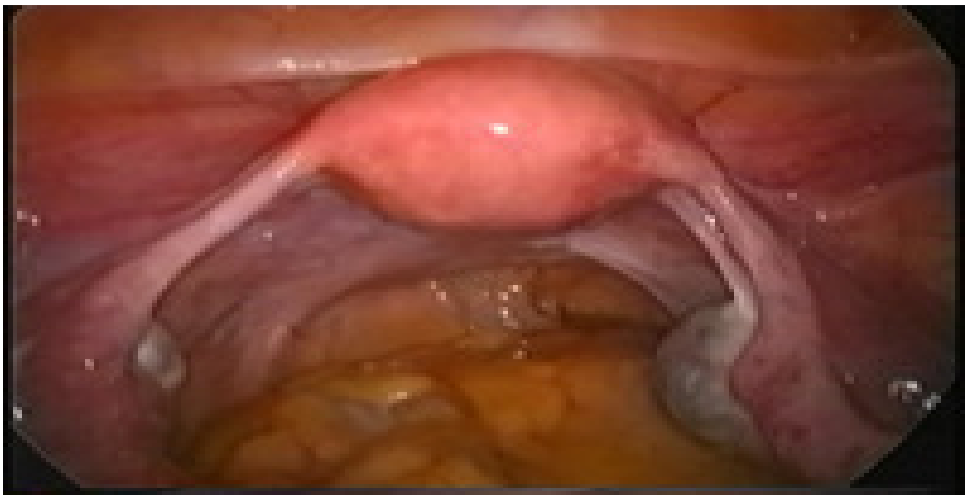
Images of sonohysterography. The uterine wall and saline in the uterine cavity are seen as black or "empty" space. A fibroid (benign tumor) is seen in the uterine cavity in the image on the right.

Uterine cavity distended with saline:

Fluid in POD:

LAPAROSCOPY:

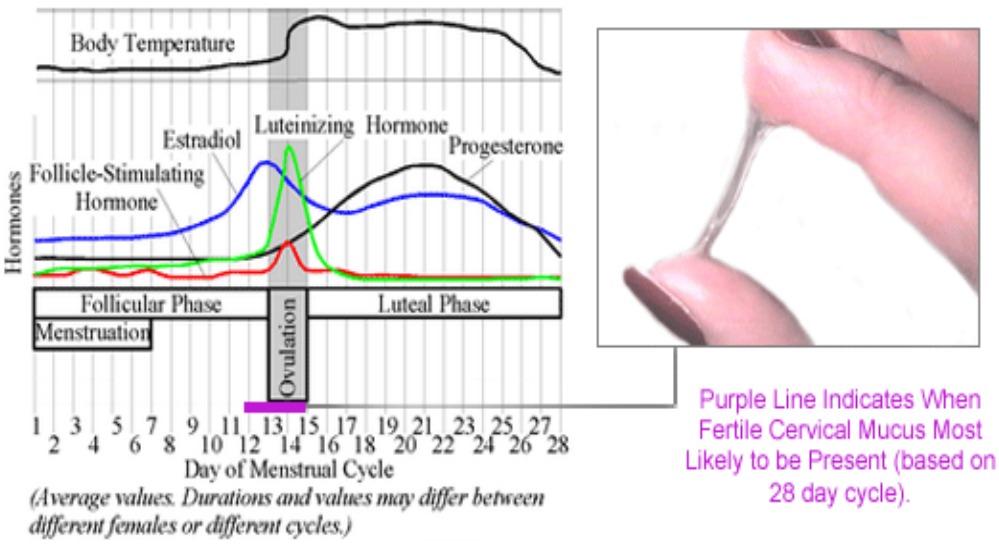
It is the gold standard technique for diagnosing peritoneal and tubal disease. It allows visualization of all pelvic organs and permits detection of periovarian and peritubal adhesions, intramural and subserosal uterine fibroids, and endometriosis. Chromopertubation involves the transcervical instillation of a dye, such as indigo carmine or methylene blue. Direct laparoscopic visualization of the dye coming through the fimbrial openings of the tubes is diagnostic of tubal patency. This technique allows assessment of the external architecture of the tubes and the visualization of the fimbria. Identified abnormalities, including pelvic adhesions, tubal blockade and endometriosis, are sometimes treated at the time of diagnosis.



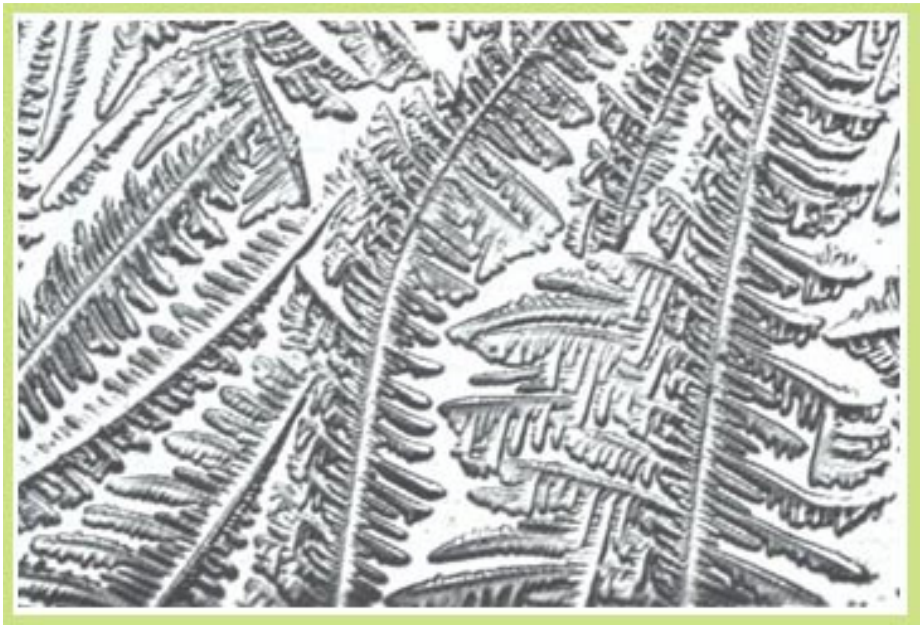
Cervical and Immunologic Factors

Postcoital Test:

Cervical factor is a factor of subfertility in about 5% of subfertile couples. The Post coital test is used to assess the number and the presence of motile sperms in the female reproductive tract after coitus, the quality of cervical mucus, and the interaction between sperm and cervical mucus. It does not yield sufficient information on sperm morphology ,its motility,sperm count, semen quality. It is performed 1 to 2 days before the anticipated time of ovulation as sufficient estrogenization of the cervical mucus is important for the interpretation of the results. Before Post Coital Test ,intercourse after 2 days of abstinence and about 2 to 12 hours is sufficient for testing purposes.



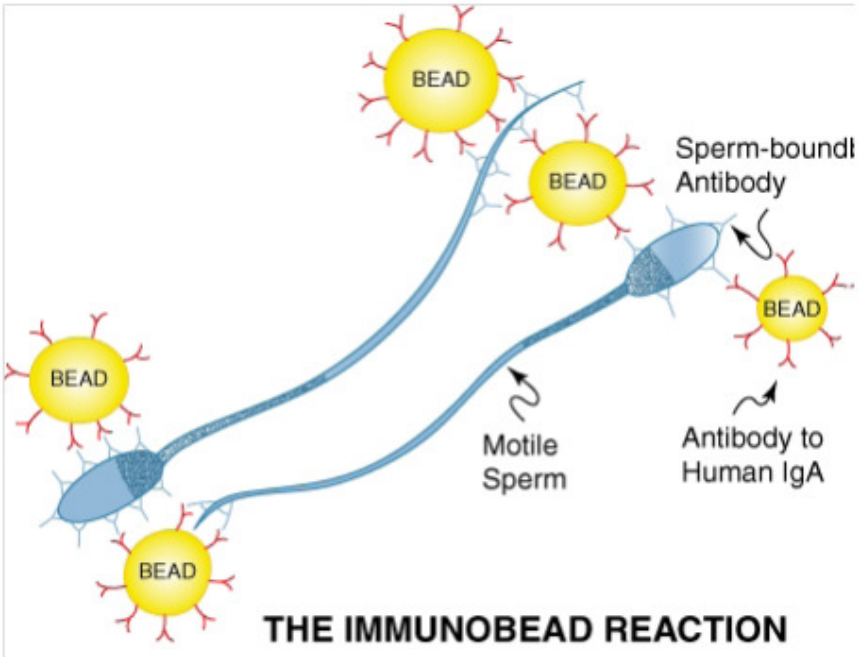
Cervical mucus can be assessed for clarity and for spinnbarkeit (i.e., stretchability), when pulled from the cervix with normal estrogen–stimulated mucus stretches upto eight to ten cm. When placed on a glass slide and covered with a cover slip, "Ferning pattern" can be assessed.



Estrogenized mucus has a characteristic ferning pattern and is clear and watery. On the other hand, progesteronized mucus is thick, opaque and lacking ferning. The number per high–power field, motility and presence of sperm, are assessed by the examination of several microscopic fields.

Few of the other causes of poor mucus quality include infection, anovulation, other anatomic factors(like prior cervical conization, cryotherapy), and the use of certain medications. Clomiphene citrate which has an antiestrogenic action on cervical glands, may have detrimental effects on cervical mucus .The presence of antisperm antibodies is suggested by observation of uniformly dead sperm.

Antisperm Antibodies



Immunobead or mixed antiglobulin reaction test: Tests for the presence of antibodies coating the sperm.

These specialized tests may be pursued to assess sperm viability, the presence of antisperm antibodies , fertilization potential (zona–free hamster oocyte test), and the effect of cervical mucus on sperm viability and function (postcoital test) The capability to mount a humoral response to sperm is present in both the male and the female which could adversely affect fertility. Antisperm antibodies (ASA) are most commonly limited to immunoglobulin G (IgG), IgM, and IgA isotypes, with each subclass having their characteristic anatomic localization. Agglutinating antibodies of the IgA class are found in seminal plasma and cervical mucus. Systemically produced IgG molecules is found in in cervical mucus, serum as well as and semen. The larger IgM antibodies which have difficulty traversing the genital tract mucosa ,are found exclusively in serum. Even in ovarian follicular fluid antisperm antibodies have been detected. In addition to subclassification by isotype, antisperm antibodies can be free, bound to sperm ,agglutinating, that is motile, or bound to sperm that is immobilized. The sperm– bound antisperm antibodies is seen to bind to different parts of the outer sperm plasma membrane. Cause is not clearly understood. In men, the exposure to sperm and their antigens to serum is shielded by blood–testis barrier . Conditions causing breaks in this barrier could activate autoimmunity Sperm complement–dependent immobilization tests –“Isojoma's” and Sperm agglutination

tests –“Kibrick's or Franklin–Dukes” have largely been replaced by the immunobead or mixed agglutination tests. The washed spermatozoa which are exposed to the labeled beads are assessed for the sperm binding. It yields specific information on both the site of binding to the involved sperm and the immunoglobulin class of the antisperm antibody. In the mixed agglutination reaction, the male's semen is mixed with human red blood cells sensitized with human IgG. The formation of mixed agglutinates with the red blood cells indicates the presence of antibody– coated spermatozoa.

Uterine Factors

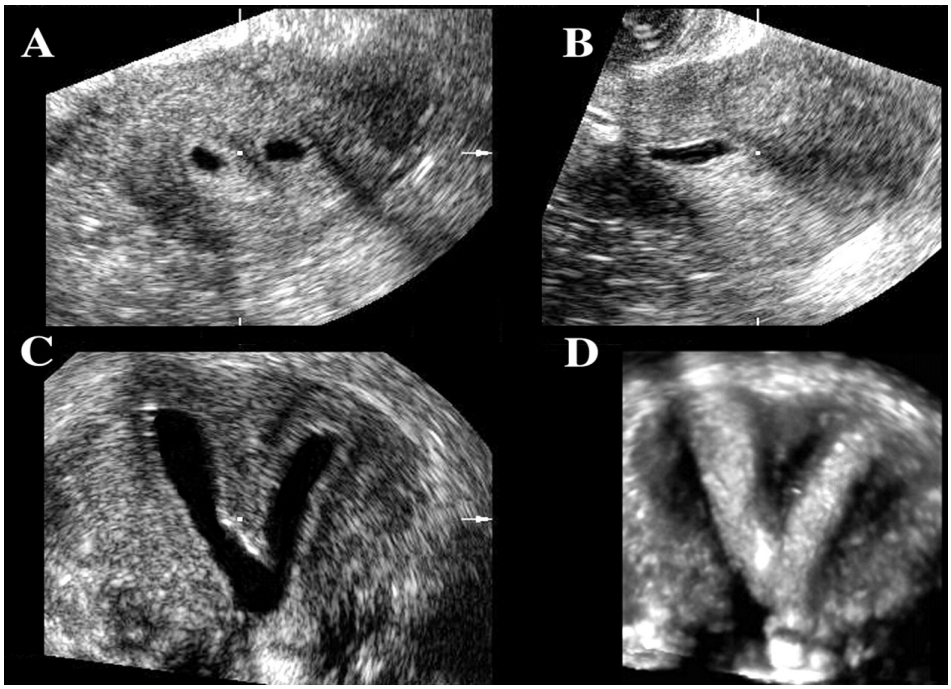
In as many as 15% of couples uterine pathologies are the cause of subfertility and in as many as 50% of subfertile patients it can be diagnosed.Lower pregnancy rates are observed in the presence of uterine cavity anomalies in patients undergoing *in vitro* fertilization.

Diagnostic Imaging for Uterine Pathology

Uterine abnormalities that have been implicated in subfertility include intrauterine adhesions, endometrial polyps, mullerian anomalies, submucous fibroids, prior exposure to *diethylstilbestrol (DES)*, and possibly luteal phase defect. Sonohysterosalpingography is known to be superior to HSG in the detection of uterine malformations, as it exactly identifies 90% of abnormalities in subfertile patients.

Sonohysterosalpingography has sensitivities of 93% and 87% in the detection of endometrial polyps and intrauterine pathology respectively. To afford a sonographic assessment of tubal status the use of contrast medium (i.e., Echovist) at the conclusion of saline sonohysterosalpingography was developed. This study is referred to as hysterosalpingo–contrast sonography, or HyCoSy.

Congenital Anomalies of the Uterus



Congenital uterine anomalies is associated with either subfertility or spontaneous pregnancy loss in the first or second trimester, and/ or late–trimester pregnancy complications. Spontaneous abortion and preterm delivery rates are highly increased in women with unicornuate, didelphic, and septate uteri at 25% to 47% and 25% to 38%, respectively.

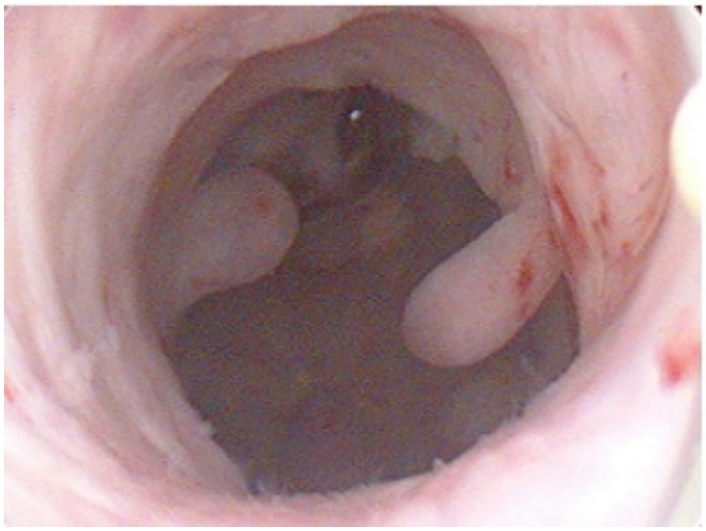
Endometrial dysfunction occurring after implantation may result in pregnancy loss, whereas dysfunction during the luteal phase may result in subfertility. Subfertility associated with most congenital uterine anomalies,with the exception of a septate uterus,is not readily amenable to surgical treatment. The risk of spontaneous abortion in women with septate uteri can be readily treated with laparoscopic–guided hysteroscopic septoplasty.Among a series of hysteroscopic metroplasties that were performed for subfertility, the overall pregnancy rate after treatment was found to be about 48% .

Acquired Abnormalities of the Uterus

Leiomyomas

Among women with subfertility and uterine leiomyomas size, number (solitary versus multiple) , location, as well as the symptoms associated with these tumors. They are not a direct cause of subfertility. Both IUI and myomectomy are reasonable treatment options for women with uterine leiomyoma and unexplained subfertility, especially if the leiomyomas are large, solitary, submucous, or distorting the uterine cavity.

Endometrial Polyps:

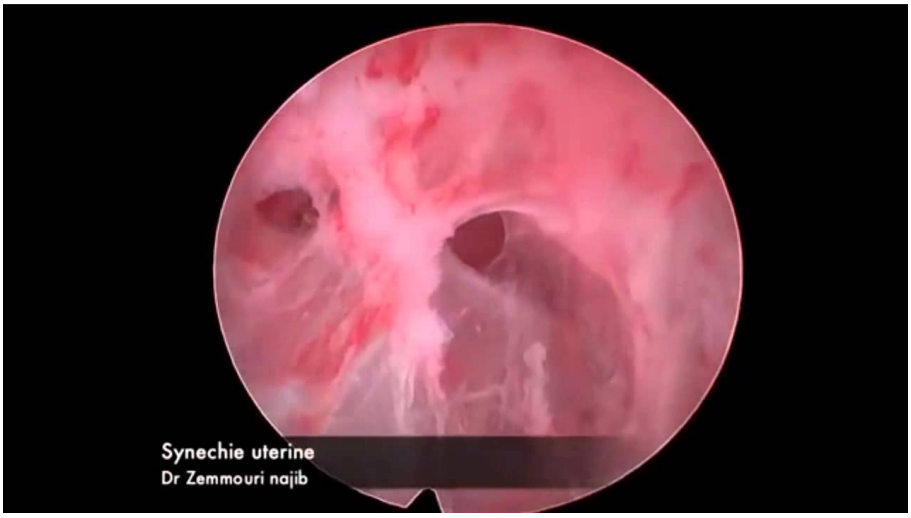


The incidence of endometrial polyps in women with subfertility is reported to be between 10% and 32%. Due to the influence of circulating estrogen on the development of endometrial polyps, among the subfertility population there may be hyperestrogenemia associated with prior cycles of controlled ovarian hyperstimulation.

Asherman Syndrome or Intrauterine Synechiae : Causes of intrauterine adhesions are

- often iatrogenic, with patient histories typically involving MTP
- D and C
- Intrauterine infection with agents such as schistosoma and mycobacteria.

In India, tuberculous endometritis is an important cause of uterine factor of subfertility. It differs from most other types of endometrial infection, and even after treatment uterine scarring and subfertility are significant sequelae. Due to intrauterine adhesions interfering with embryo implantation, severe forms of Asherman syndrome have been associated with spontaneous abortion, amenorrhea, menstrual irregularities, and recurrent pregnancy loss.

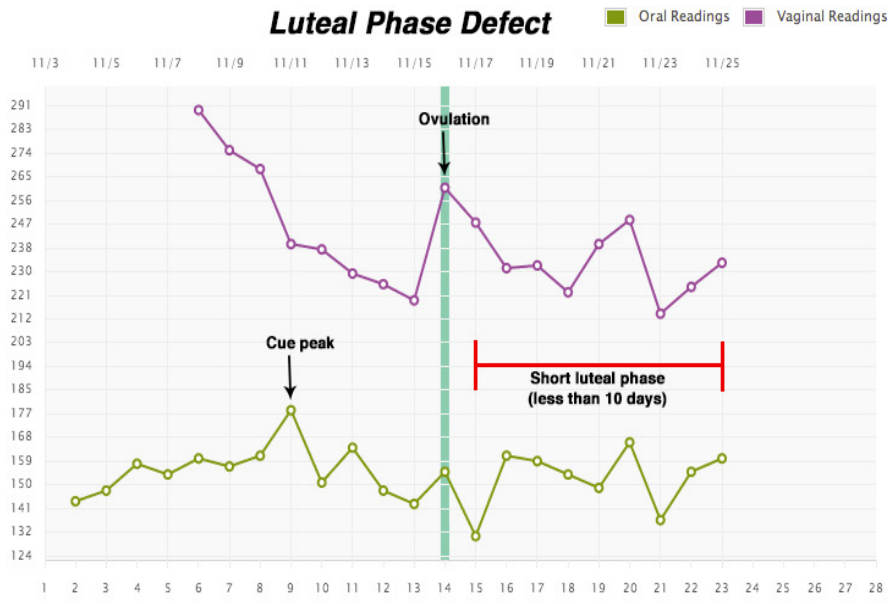


Intrauterine synechiae is managed by hysteroscopic resection adhesions may be prevented by estrogen therapy alone or combining with placement of an intrauterine device such as a copper-T or pediatric Foley catheter. Estrogens rapidly rebuild the endometrial lining after surgery and thus prevent the development of scar tissue. The surgical management of intrauterine adhesions is found to be very effective, with

pregnancy rates as high as 80% among patients who were treated with mild to moderate disease.

Disorders of Endometrial Function and Luteal–phase Defect

Luteal–phase defect is said to be present when two endometrial biopsies show a delay of 2 days or more beyond the actual day of the cycle in the histologic development of the endometrium. Luteal Phase Defect can also be characterized by delayed endometrial maturation,histologic asynchrony between endometrial epithelial and stromal compartments. Other factors which may be responsible for such histologic alterations, including hyperprolactinemia, inadequate follicular development, inadequate luteinising hormone or follicle stimulating hormone secretion, and inadequate progesterone production by the corpus luteum .



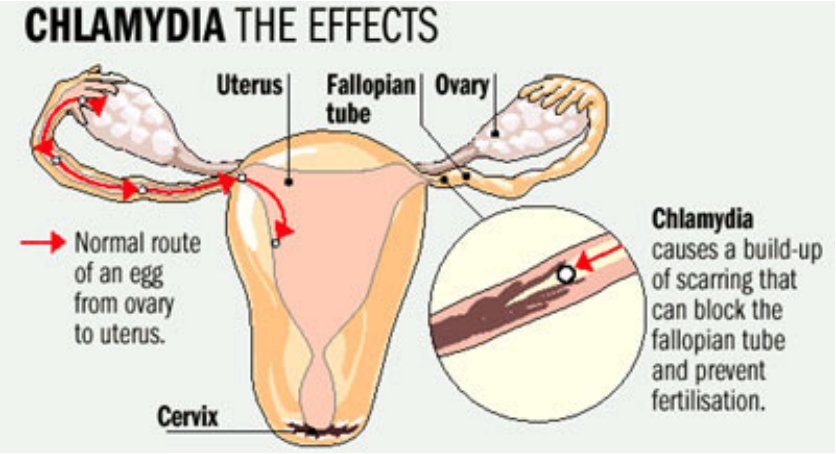
Though some studies have advocated the use of a threshold serum progesterone level in luteal–phase, data associating either single or summed midluteal–phase serum progesterone levels with proposed luteal phase abnormalities are lacking due to the characteristic pulsatile secretion of progesterone. Studies of IVF and embryo transfer have indicated that the implantation window lasts from day 5.5 to day 9.5 postovulation.

Luteal–phase defect is said to adversely affect endometrial receptivity to blastocyst implantation because of the formation of a nonreceptive endometrial environment as a result of an aberrant molecular profile within the endometrium. To this end, both the presence of pinopodes and molecular markers, such as the expression of integrins, have been proposed as indicators of uterine receptivity . Although αγβ3 integrin expression and pinopode formation appear to be more reliable markers of the implantation window than histologic dating, the clinical utility of testing for their presence is questionable because of the significant intercycle variability and poor reproducibility in their expression. The value of αγβ3 integrin expression as a predictor of pregnancy outcome has been recently refuted in a controlled ovarian hyperstimulationort of 100 consecutive subfertile patients who underwent mid– and late luteal, endometrial biopsy–based histologic dating and immunohistochemistry for αvβ3 expression. Current treatments for presumed LPD in subfertile patients are empirical and reflect the

hypothesis that progesterone insufficiency is causal. Treatment, therefore, involves the administration of vaginal micronized (400–600 mg/day) or intramuscular *progesterone* (50–100 mg/day) beginning 3 days after documentation of an luteinising hormone surge. After ART with protocols involving a GnRH agonist, supplementation via vaginal administration may be preferred. Progesterone supplementation may be continued till the 1st day of the next menstrual cycle or negative serum quantitative hCG value. If a patient becomes pregnant while on therapy, progesterone is continued until eight to ten weeks of gestation. Studies supporting progesterone supplementation for LPD have reported improved pregnancy rates after intervention. However, these studies have been small and poorly controlled with varied diagnostic criteria.

Infectious Factors

The two potential pathogens are: *Chlamydia trachomatis* and *Mycoplasma* species. The association of chlamydia and PID is well established.



Chlamydia is the predominant pathogen detected in nearly 20% cases of acute salpingitis. Chlamydia may also produce some asymptomatic infection in the female genital tract, and hence some women experience silent tubal infection. Inspite of just a few if any symptoms, these infections may result in significant tubal damage. There may be a possible link between infection and subfertility as evidenced by the prevalence of positive chlamydial cultures among subfertile patients than among controls.There was not much of a difference in reproductive outcomes of IVF. In a study of 771 patients undergoing egg retrieval the incidence of bacterial vaginosis was 25%(as part of IVF treatment) Although pregnancy rates were not affected, bacterial vaginosis patients there was a significantly higher risk for spontaneous abortion even after adjusting for maternal age, smoking history, history of recurrent miscarriage, and the presence of polycystic ovaries, and history of previous live birth. It is also not known if treatment of bacterial vaginosis before or during IVF treatment would change the rate of spontaneous abortion. Mycoplasma species are pleuropneumonia like organisms. Both Ureaplasma urealyticum *and* Mycoplasma hominis are recovered from the cervical mucus and semen of the couples. There is a high rate of mycoplasma infection among subfertile couples than among fertile couples.

Systemic Illness

In general, any severe systemic illness, leading to disruption of the hypothalamic–pituitary–ovarian axis such as liver failure, renal failure, or metastatic cancer, can cause subfertility. Even after a period of anovulation, because sporadic ovulation is always possible, use of contraception is advised if pregnancy is not desired. If a patient with severe systemic illness wishes to conceive, careful preconceptional assessment and counselling is advisable because the risks of fertility treatment and pregnancy can be substantial. The association of antiphospholipid antibodies, particularly anticardiolipin antibodies and the lupus anticoagulant, with recurrent pregnancy loss led to the investigation of a role for these antibodies in subfertility. These antibodies are more prevalent in the subfertility population. However, the presence of antiphospholipid antibodies has not been found to adversely affect IVF outcomes in a prospective study or in a meta–analysis of seven studies. These findings do not support a role for the routine testing of antiphospholipid antibodies in the subfertility evaluation.

Unexplained Subfertility

If the basic evaluation reveals normal semen parameters, evidence of ovulation, patent fallopian tubes, and no other obvious cause of subfertility, the couple is diagnosed with unexplained subfertility. This diagnosis accounts for up to 30% of couples with subfertility. In six

randomized studies of couples with unexplained subfertility, the cycle fecundity in untreated controls was 3.8%, significantly lower than the 25% fecundity rate observed in normal fertile couples.

MANAGEMENT:

TUBAL RECONSTRUCTION:

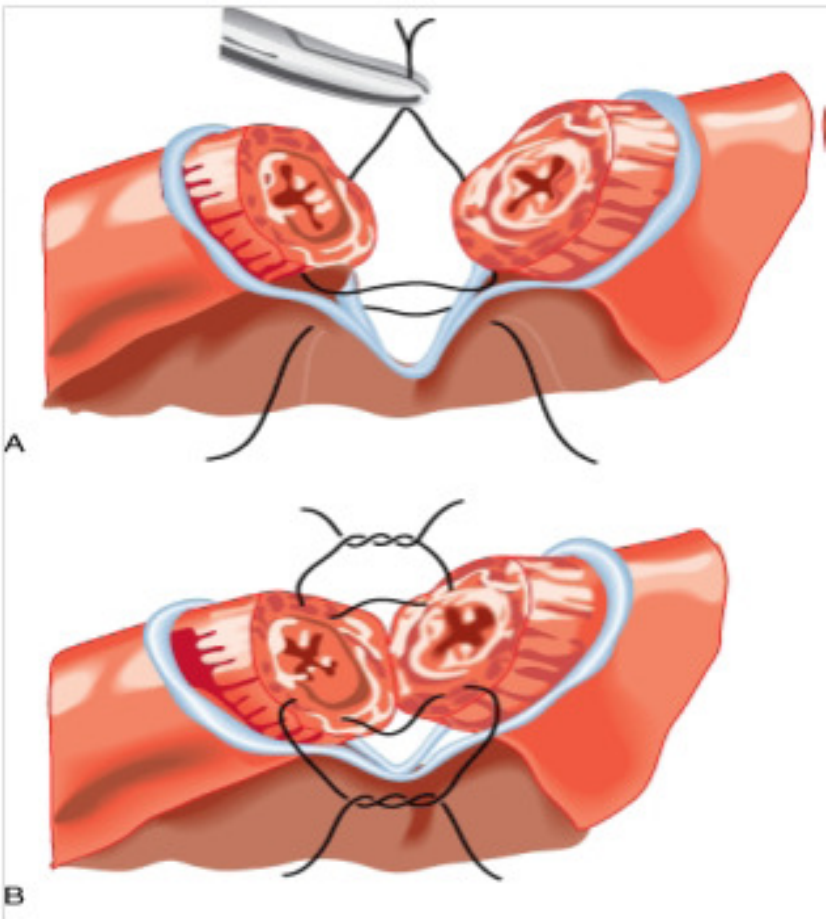
Proximal Tubal Obstruction:

Tubal obstruction options include surgical reanastomosis, hysteroscopic cannulation, and neosalpingostomy. Reproductive surgery is an important option or complement to ART for many couples though there have been considerable increases in the success rates of ART. Proximal occlusion extending to the interstitial segment which cannot be treated with cannulation is best treated with IVF. Surgical reanastomosis of fallopian tube segments.

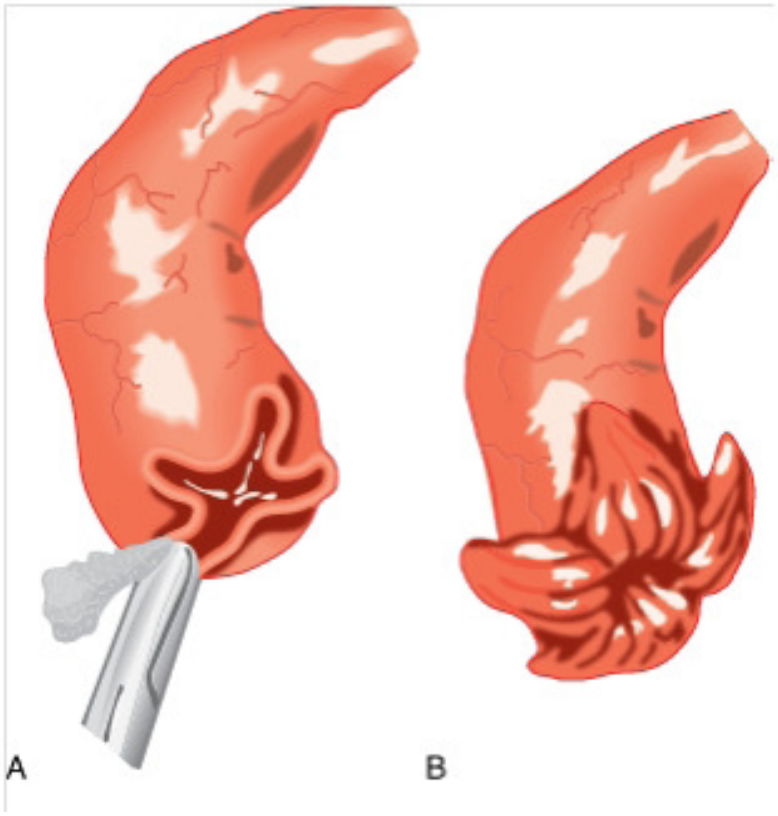
A. The scarred portion of the tube is excised until nonfibrotic tubal tissues are reached.
B. The mesosalpinx is then re-approximated with interrupted stitches using 6-0 delayed-absorbable suture.
C. The tubal muscularis is reapproximated with single stitches in each quadrant using 7-0 delayed-absorbable suture.
D. Tubal serosa is closed with interrupted or running 6-0 delayed-absorbable suture.

Distal Tubal Obstruction

Following pelvic inflammatory disorders, normal fimbrial anatomy may be destroyed or fimbria may be encased by concomitant adnexal adhesions. In these cases, neosalpingostomy can be performed by mini-laparotomy or laparoscopy.



These women who desire neosalpingostomy should be counseled about the risk of ectopic pregnancy.

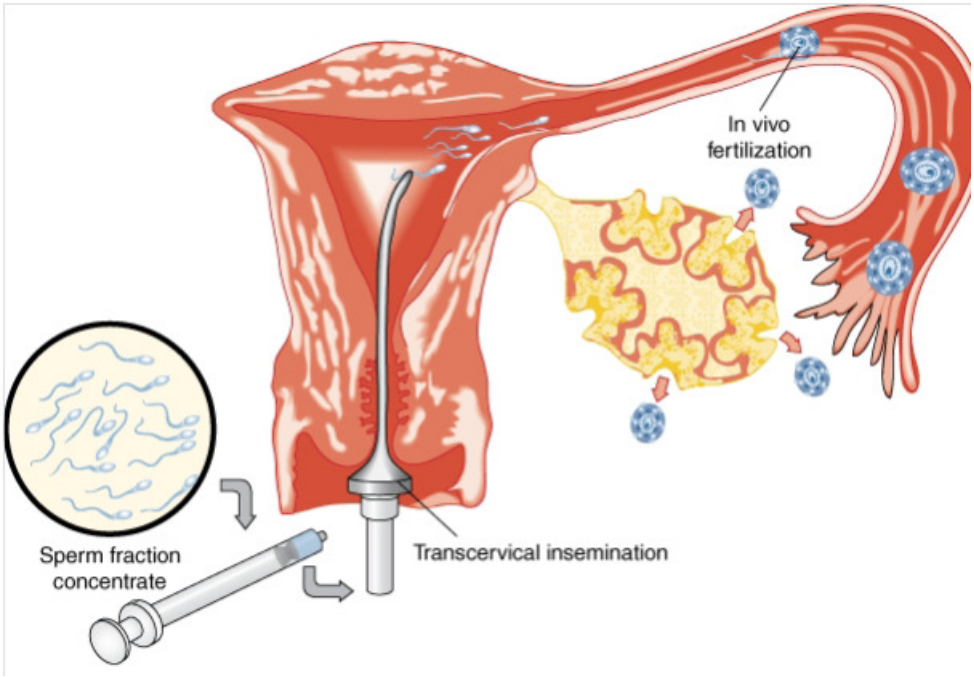


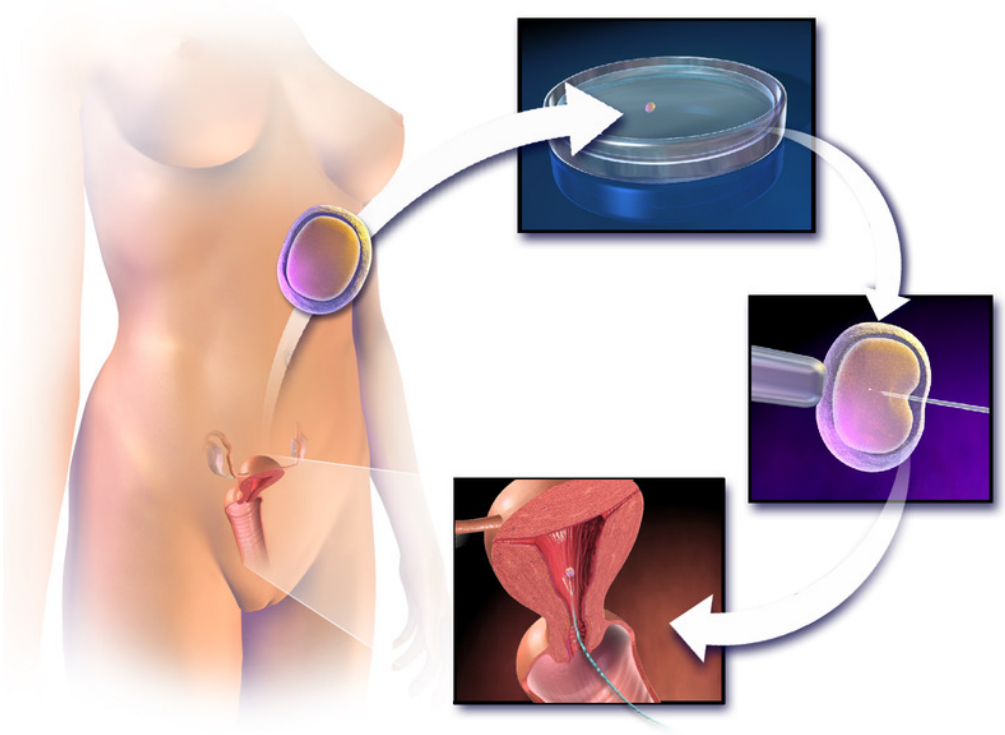
Neosalpingostomy:

- The distal ends of the clubbed fallopian tubes are opened with electric or laser energy.
- The endosalpinx is then everted using Cuff or Bruhat technique.

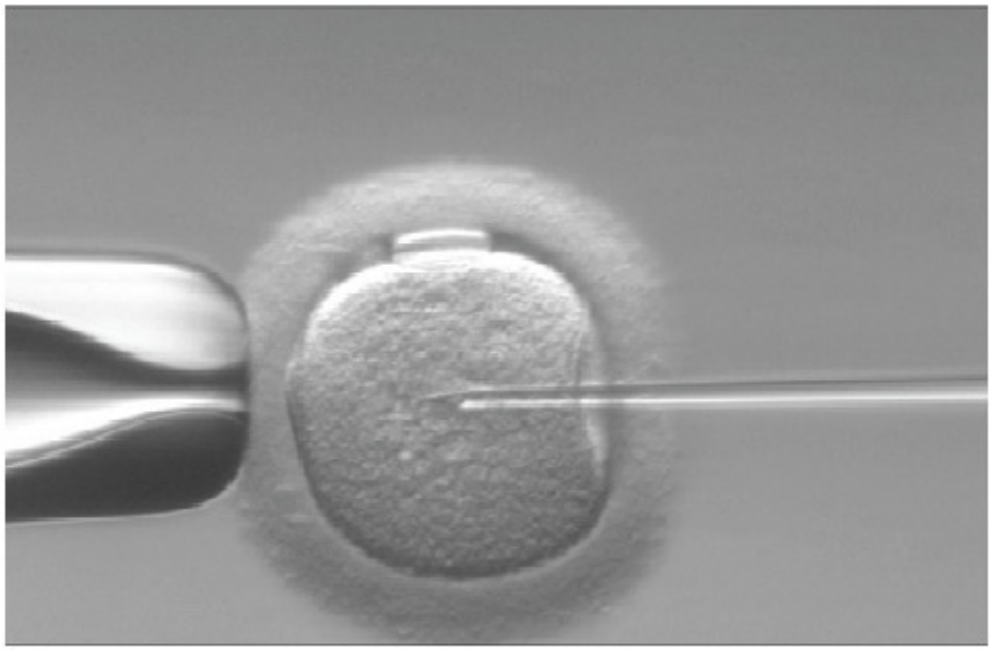
INTRAUTERINE INSEMINATION:

Intracervical insemination is the technique of injection of unwashed or raw semen into the cervix with syringe. Semen used in intrauterine insemination should be fresh, raw or frozen. When an ovum is released, semen is introduced into the woman's vagina, uterus or cervix. If fresh semen is used, it should be allowed to liquefy before inserting it into the syringe, or the syringe can be back-loaded. Care is taken when inserting the syringe, so that the tip is as close to the entrance to the cervix as possible and the syringe is inserted more accurately through the open speculum .





OTHER TECHNIQUES OF ASSISTED REPRODUCTIVE TECHNIQUES:

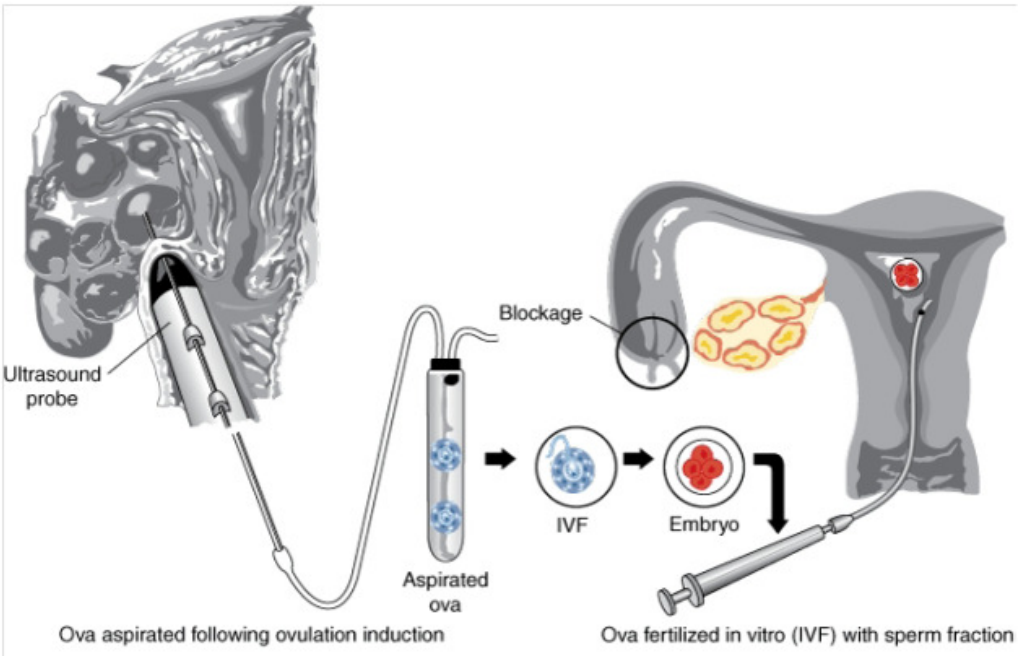


ICSI AND IMSI:

ICSI : It is usually used to overcome male subfertility issues, but can also be used in conditions where ovum is not easily penetrated by sperm.It can also be used in teratozoospermia, as after fertilisation, abnormal sperm morphology will not influence blastocyst development

or its morphology. The advantage of this procedure is that even with severe teratozoospermia, microscopy would still detect the few sperm cells that have a "normal" morphology, allowing for optimal success rate.

Intracytoplasmic morphologically selected sperm injection (IMSI) is a variation of ICSI which uses a higher-powered microscope to select sperm. This option allows embryologists to look at the sperm in greater detail (including the nucleus which contains the sperm's genetic material). Some studies suggest that using this technique selects better quality sperm and results in higher pregnancy rates and lower miscarriage rates compared to conventional ICSI. The shape of the sperm (morphology) is important in diagnosing male fertility problems and in predicting fertilization and pregnancy outcomes and studies have shown that selecting better shaped sperm improves ICSI outcomes. In conventional ICSI, the embryologist selects the most normal-looking motile sperm using a microscope that magnifies the sample up to 400 times. The high power light microscope (enhanced by digital imaging) used for IMSI magnifies the sperm sample over 6000 times. This allows the embryologist to detect subtle structural alterations in sperm that a normal microscope could not detect. Sperm are then selected which have the most normally-shaped nuclei.



In vitro fertilization (IVF):

Here, at the time of ovulation, following ovulation induction,eggs from the ovaries are harvested and are fertilized in vitro.This fertilized egg develops to the blastocyst stage and are drawn up into a syringe and placed inside the endometrial cavity.

AIM OF THE STUDY

- ✓ To compare the accuracy of two methods of assessment of tubal patency in cases of primary and secondary subfertility.
- ✓ To find out whether Sonosalpingography, which is a less invasive method, can be used for assessment of tubal factor in cases of primary and secondary subfertility initially instead of Diagnostic laparoscopy with chromopertubation which is associated with significant morbidity and even some mortality.

PERIOD OF STUDY: September 2013 to September 2014

PLACE OF STUDY:

Pregynaec ward, Ultrasound room and Gynaec OT Department of obstetrics and Gynaecology. Govt. Kilpauk Medical College Hospital, Chennai

MATERIALS AND METHODS

INCLUSION CRITERIA:

All patients with primary/secondary subfertility attending gynaec OPD in the age group of 18 to 40 years, not with below mentioned exclusion criteria.

EXCLUSION CRITERIA:

- All established cases of hydrosalpinx as the tubal flow may give a false impression of tubal patency in SSG.
- Pregnancy and PID
- All medical contraindications for Diagnostic laparoscopy

Sample size:

Sample Size for Frequency in a Population

Population size(for finite population correction factor or fpc)(*N*):	700
Hypothesized % frequency of outcome factor in the population (*p*):	15%+/-5
Confidence limits as % of 100(absolute +/- %)(*d*):	5%
Design effect (for cluster surveys-*DEFF*):	1

Sample Size (*n*) for Various Confidence Levels

CONFIDENCE LEVEL (%)	**Sample Size**
95%	154
80%	75
90%	116
97%	180
99%	229
99.9%	309
99.99%	368

Equation

Sample size n = $[DEFF*Np(1-p)]/ [(d^2/Z^2_{1-\alpha/2}*(N-1)+p*(1-p)]$

In this scenario, Total number of sub-fertility patients for 1 year in Govt. KMCH is about 700 .The prevalence for tubal patency is 15 %. By based on the above assumptions, we made calculations and the sample size is 154 for 95 % Confidence Interval.

By taking the lost to follow up and other causes, the **sample size is 170.**

Method:

The study is conducted in the Dept. of Obstetrics and Gynaecology, Govt. KMCH among women with primary/secondary subfertility attending Gynaec OP .After getting informed consent for the study, patients were evaluated by

History taking

General examination

Pelvic examination.

- SSG is done on 7th or 8th day of the menstrual cycle, in the USG room of Gynaec OPD premises.

- An informed consent is taken. A transvaginal ultrasound is performed prior to SIS to look for any endometrial polyp and presence of fluid in the pouch of douglas (POD). The vulva and vagina were cleaned with antiseptic solution. Sims speculum is introduced and the anterior lip of cervix is held with vulsellum. A sterile 5F- 8F paediatric foleys catheter is inserted into the uterine cavity. The catheter is prefilled with saline prior to insertion to minimise artefact. The catheter is repositioned so as to snugly fit into the cervical canal to prevent the back flow of saline. The speculum is removed and continuous intravenous drip of normal saline is connected to the catheter. Once

adequate distension of uterine cavity is achieved, the cavity is evaluated for presence of any abnormality. Presence of fluid in POD after SIS which is previously absent on ultrasonography is also taken as a sign of tubal patency. At the end of the procedure retrograde leakage, pain and time taken for the procedure are also noted.

- Diagnostic laparoscopy with chromopertubation was performed under general anesthesia on the following day to evaluate pelvic pathology and tubal patency. This was performed by methylene blue dye injection through a cannula. If the methylene blue dye could pass through the distal end of fimbria at least one side, it represented tubal patency (positive test). Whereas the dye could not pass through the distal end of both fimbriae, it represented tubal occlusion (negative test).

- The data are subsequently analyzed to compare the results of the two procedures and to find out the accuracy of Sonosalpingography in comparison with the Diagnostic laparoscopy.

BENEFITS OF THE STUDY:

Evaluates the efficacy of SSG as an alternative to Diagnostic laparoscopy in assessment of tubal patency in patients with primary/secondary subfertility.

SSG offers a much less invasive method of diagnosing tubal pathology while maintaining a high sensitivity and specificity similar to that of laparoscopic chromopertubation.

Moreover SSG can be done for patients who have bronchial asthma or cardiac problems and are temporarily unfit for Surgery.

RESULTS

TUBAL PATENCY:

McNemar test:

Classification A	TUBAL_PATENCY_LAP TUBAL PATENCY LAP
Classification B	TUBAL_PATENCY_SSG TUBAL PATENCY SSG

	Classification A		
Classification B	0	1	
0	18	5	23 (13.5%)
1	2	145	147 (86.5%)
	20 (11.8%)	150 (88.2%)	170

Difference	1.76%
95% CI	-1.73 to 3.82

Exact probability (binomial distribution)

Significance	**P = 0.4531**

Diagnostic test

	Disease Present	Disease Absent	
Test Positive	145	2	147
Test Negative	5	18	23
	150	20	

Results

Sensitivity	96.67%	92.39% to 98.91%
Specificity	90.00%	68.30% to 98.77%
Positive Likelihood Ratio	9.67	2.59 to 36.01
Negative Likelihood Ratio	0.04	0.02 to 0.09
Disease prevalence	88.24%	82.42% to 92.66%
Positive Predictive Value	98.64%	95.17% to 99.83%
Negative Predictive Value	78.26%	56.30% to 92.54%

Diagnostic or Screening Test Evaluation:

Single Table Analysis

	Positive	Negative	Total
Positive	145	2	147
Negative	5	18	23
	150	20	170

RESULTS:

Parameter	Estimate	Lower - Upper 95% CIs	Method
Sensitivity	96.67%	(92.43, 98.57[1])	Wilson Score
Specificity	90%	(69.9, 97.21[1])	Wilson Score
Positive Predictive Value	98.64%	(95.18, 99.63[1])	Wilson Score
Negative Predictive Value	78.26%	(58.1, 90.34[1])	Wilson Score
Diagnostic Accuracy	95.88%	(91.75, 97.99[1])	Wilson Score

CONCLUSION:

It was found that SSG has a sensitivity of 97% and specificity of 90% and a diagnostic accuracy of 96% when compared to diagnostic laparoscopy in detection of tubal patency.

SSG detected 23 cases of tubal block of which 5 cases were ruled out as patent by D'LAP.2 cases detected as patent tubes by SSG had tubal block in D'LAP.

ENDOMETRIOSIS:

McNemar test:

Classification A	ENDOMETRIOSIS__LAP ENDOMETRIOSIS LAP
Classification B	ENDOMETRIOSIS__SSG ENDOMETRIOSIS SSG

	Classification A		
Classification B	0	1	
0	166	4	170 (100.0%)
1	0	0	0 (0.0%)
	166 (97.6%)	4 (2.4%)	170

Difference	2.35%
95% CI	-0.48 to 2.35

Exact probability (binomial distribution)

Significance	**P = 0.1250**

Diagnostic test

	Disease Present	Disease Absent	
Test Positive	0	0	0
Test Negative	4	166	170
	4	166	

Results

Sensitivity	0.00%	0.00% to 60.24%
Specificity	100.00%	97.80% to 100.00%
Positive Likelihood Ratio		
Negative Likelihood Ratio	1.00	1.00 to 1.00
Disease prevalence	2.35%	0.64% to 5.91%
Positive Predictive Value		
Negative Predictive Value	97.65%	94.09% to 99.36%

CONCLUSION:

It was found that SSG has a low sensitivity in detecting endometriosis compared to diagnostic laparoscopy.

ADHESIONS:

McNemar test

Classification A	ADHESIONS__LAP ADHESIONS LAP
Classification B	ADHESIONS__SSG ADHESIONS SSG

	Classification A		
Classification B	0	1	
0	159	11	170 (100.0%)
1	0	0	0 (0.0%)
	159 (93.5%)	11 (6.5%)	170

Difference	6.47%

95% CI	2.78 to 6.47

Exact probability (binomial distribution)

Significance	**P = 0.0010**

Diagnostic test

	Disease Present	Disease Absent	
Test Positive	0	0	0
Test Negative	11	159	170
	11	159	

Results

Sensitivity	0.00%	0.00% to 28.49%
Specificity	100.00%	97.71% to 100.00%
Positive Likelihood Ratio		
Negative Likelihood Ratio	1.00	1.00 to 1.00
Disease prevalence	6.47%	3.27% to 11.28%
Positive Predictive Value		
Negative Predictive Value	93.53%	88.72% to 96.73%

CONCLUSION:

Considered to be one of the drawbacks of this study was the inability to detect peritubal adhesions associated with PID/Genital TB. Adhesions were detected in 11 of 170 patients during diagnostic laparoscopy.

FIBROID/POLYP:

McNemar test

Classification A	FIBROID_POLYP__LAP FIBROID_POLYP LAP
Classification B	FIBROID_POLYP__SSG FIBROID_POLYP SSG

	Classification A		
Classification B	0	1	
0	164	2	166 (97.6%)
1	0	4	4 (2.4%)
	164 (96.5%)	6 (3.5%)	170

Difference	1.18%
95% CI	2.78 to 6.47

Exact probability (binomial distribution)

Significance	**P = 0.5000**

Diagnostic test

	Disease Present	Disease Absent	
Test Positive	4	0	4
Test Negative	2	164	166
	6	164	

Results

Sensitivity	66.67%	22.28% to 95.67%
Specificity	100.00%	97.78% to 100.00%
Positive Likelihood Ratio		
Negative Likelihood Ratio	0.33	0.11 to 1.03
Disease prevalence	3.53%	1.31% to 7.52%
Positive Predictive Value	100.00%	39.76% to 100.00%
Negative Predictive Value	98.80%	95.72% to 99.85%

CONCLUSION:

DIAGNOSTIC ACCURACY: 99%.

One of the interesting findings noted in the study was the detection of 1 endometrial polyp and 1 submucous fibroid which were missed in diagnostic laparoscopy.2 posterior wall fibroids were missed in SSG and detected during D'LAP. The submucous polyp detected was subsequently removed through hysteroscopy.

PCOD:

McNemar test:

Classification A	PCOD__LAP PCOD LAP
Classification B	PCOD__SSG PCOD SSG

	Classification A		
Classification B	0	1	
0	102	4	106 (62.4%)
1	0	64	64 (37.6%)
	102 (60.0%)	68 (40.0%)	170

Difference	2.35%

95% CI	-0.48 to 2.35

Exact probability (binomial distribution)

Significance	**P = 0.1250**

Diagnostic test

	Disease Present	Disease Absent	
Test Positive	64	0	64
Test Negative	4	102	106
	68	102	

Results

Sensitivity	94.12%	85.62% to 98.37%
Specificity	100.00%	96.45% to 100.00%
Positive Likelihood Ratio		
Negative Likelihood Ratio	0.06	0.02 to 0.15
Disease prevalence	40.00%	32.58% to 47.78%
Positive Predictive Value	100.00%	94.40% to 100.00%
Negative Predictive Value	96.23%	90.62% to 98.96%

CONCLUSION:

DIAGNOSTIC ACCURACY: 97.65%.

Almost one half of the patients had associated PCOD which was detected by both SSG and D'LAP. This re-emphasises the fact that obesity, lifestyle and eating habits have significantly caused a raise in the incidence of subfertility. 18 cases of PCOS underwent ovarian drilling along with D'LAP.

TUBOOVARIAN MASS:

McNemar test:

Classification A	TUBOOVARIAN_MASS __LAP TUBOOVARIAN_MASS LAP
Classification B	TUBOOVARIAN_MASS __SSG TUBOOVARIAN_MASS SSG

	Classification A		
Classification B	0	1	
0	163	0	163 (95.9%)
1	0	7	7 (4.1%)
	163 (95.9%)	7 (4.1%)	170

Diagnostic test

	Disease Present	Disease Absent	
Test Positive	7	0	7
Test Negative	0	163	163
	7	163	

Results

Sensitivity	100.00%	59.04% to 100.00%
Specificity	100.00%	97.76% to 100.00%
Positive Likelihood Ratio		
Negative Likelihood Ratio	0.00	
Disease prevalence	4.12%	1.67% to 8.30%
Positive Predictive Value	100.00%	59.04% to 100.00%
Negative Predictive Value	100.00%	59.04% to 100.00%

CONCLUSION:

DIAGNOSTIC ACCURACY: 100%.

There was 7 cases of associated tubo-ovarian mass detected during SSG and confirmed through LAP.3 of the cases underwent laparoscopic cystectomy along with chromopertubation.

BASED ON THE TYPE OF SUBFERTILITY:

Of the 23 tubal blocks detected by SSG, 14 were primary and 9 were secondary subfertility cases.

Of the actual 20 tubal blocks detected by D'Lap, 12 were primary and 8 were secondary subfertility cases.

TUBAL PATENCY SSG * TYPE

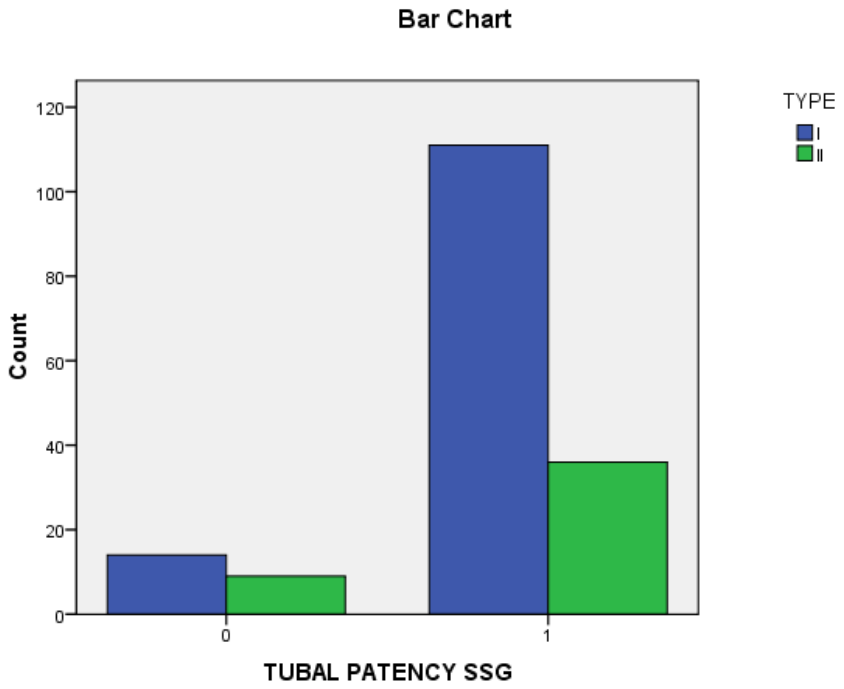
			TYPE		
			I	II	TOTAL
TUBAL PATENCY SSG	0	Count	14	9	23
		% within TUBAL PATENCY SSG	60.9%	39.1%	100.0%
		% within TYPE	11.2%	20.0%	13.5%
		% of Total	8.2%	5.3%	13.5%
	1	Count	111	36	147
		% within TUBAL PATENCY SSG	75.5%	24.5%	100.0%
		% within TYPE	88.8%	80.0%	86.5%
		% of Total	65.3%	21.2%	86.5%
	Total	Count	125	45	170
		% within TUBAL PATENCY SSG	73.5%	26.5%	100.0%
		% within TYPE	100.0%	100.0%	100.0%
		% of Total	73.5%	26.5%	100.0%

CHI SQUARE TESTS

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.190[a]	1	.139		
Continuity Correction[b]	1.503	1	.220		
Likelihood Ratio	2.046	1	.153		
Fisher's Exact Test				.202	.112
N of Valid Cases	170				

Bar chart showing distribution based on the type of subfertility.

Shade blue indicates primary and shade green indicates secondary subfertility.

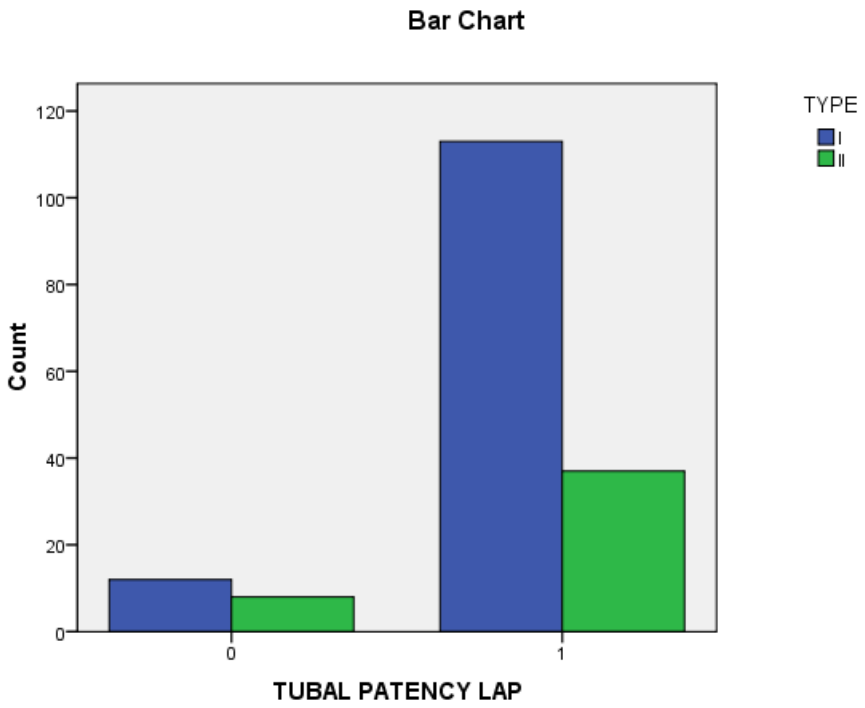


TUBAL PATENCY LAP * TYPE

			TYPE		
			I	II	TOTAL
TUBAL PATENCY LAP	0	Count	12	8	20
		% within TUBAL PATENCY LAP	60.0%	40.0%	100.0%
		% within TYPE	9.6%	17.8%	11.8%
		% of Total	7.1%	4.7%	11.8%
	1	Count	113	37	150
		% within TUBAL PATENCY LAP	75.3%	24.7%	100.0%
		% within TYPE	90.4%	82.2%	88.2%
		% of Total	66.5%	21.8%	88.2%
	Total	Count	125	45	170
		% within TUBAL PATENCY LAP	73.5%	26.5%	100.0%
		% within TYPE	100.0%	100.0%	100.0%
		% of Total	73.5%	26.5%	100.0%

CHI SQUARE TESTS

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.132[a]	1	.144		
Continuity Correction[b]	1.417	1	.234		
Likelihood Ratio	1.980	1	.159		
Fisher's Exact Test				.177	.119
N of Valid Cases	170				



CONCLUSION :

Based on the assessment of tubal patency among type 1 and type 2 subfertility, it was found that tubal block detected by SSG among type 1 and 2 subfertility were 61% and 39 % respectively as compared to LAP where it was 60% and 40% respectively.

BASED ON THE AGE GROUP:

The following bar charts show the age distribution of the findings.

Group 1 shaded blue indicates age groups less than 25years.

Group 2 shaded green indicates age group between 25-30years.

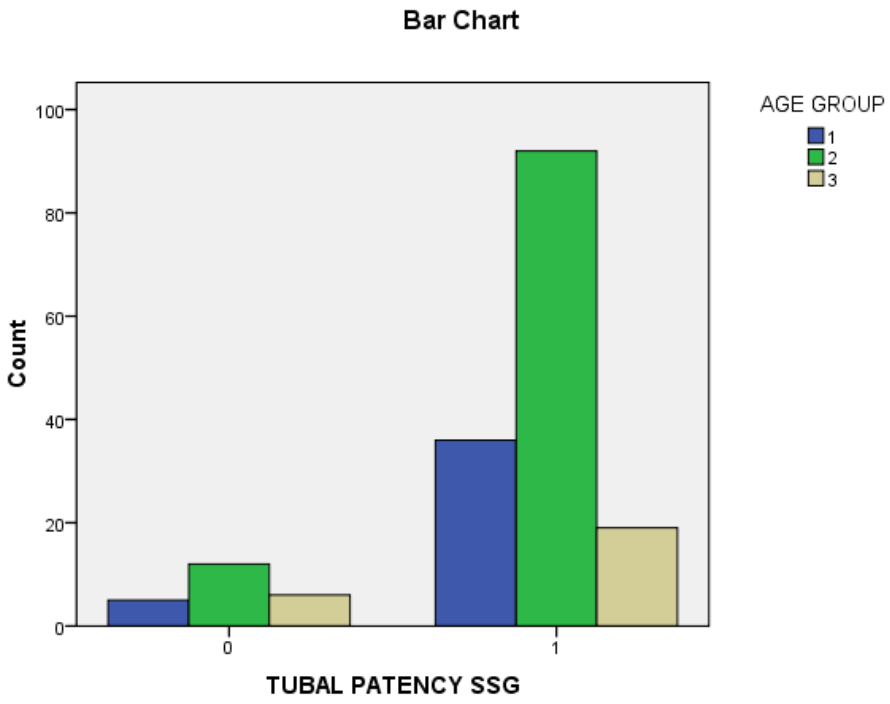
Group 3 shaded grey indicates age group over 30years.

TUBAL PATENCY SSG * AGE GROUP:

			Age groups		
			1	2	3
TUBAL PATENCY SSG	0	Count	5	12	6
		% within TUBAL PATENCY SSG	21.7%	62.6%	12.9%
		% within AGE GROUP	12.2%	11.5%	24.0%
		% of Total	2.9%	7.1%	3.5%
	1	Count	36	92	19
		% within TUBAL PATENCY SSG	24.5%	52.2%	26.1%
		% within AGE GROUP	87.8%	88.5%	76.0%
		% of Total	21.2%	54.1%	11.2%
	Total	Count	41	104	25
		% within TUBAL PATENCY SSG	24.1%	61.2%	14.7%
		% within AGE GROUP	100.0%	100.0%	100.0%
		% of Total	24.1%	61.2%	14.7%

CHI SQUARE TESTS:

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.758[a]	2	.252
Likelihood Ratio	2.406	2	.300
Linear-by-Linear Association	1.320	1	.251
N of Valid Cases	170		

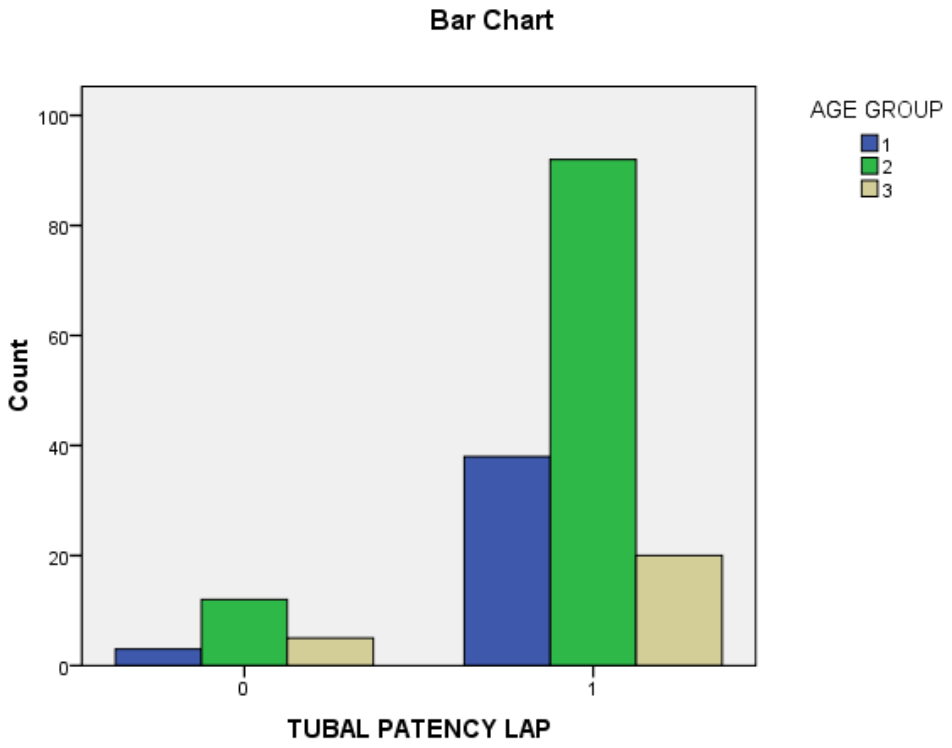

Bar Chart
AGE GROUP
1
2
3
Count
100
80
60
40
20
0
0
1
TUBAL PATENCY SSG

TUBAL PATENCY LAP * AGE GROUP

			Age groups		
			1	2	3
TUBAL PATENCY LAP	0	Count	3	12	5
		% within TUBAL PATENCY LAP	15.0%	60.0%	25.0%
		% within AGE GROUP	7.3%	11.5%	20.0%
		% of Total	1.8%	7.1%	2.9%
	1	Count	38	92	20
		% within TUBAL PATENCY LAP	25.3%	61.3%	13.3%
		% within AGE GROUP	92.7%	88.5%	80.0%
		% of Total	22.4%	54.1%	11.8%
	Total	Count	41	104	25
		% within TUBAL PATENCY LAP	24.1%	61.2%	14.7%
		% within AGE GROUP	100.0%	100.0%	100.0%
		% of Total	24.1%	61.2%	14.7%

CHI SQUARE TESTS:

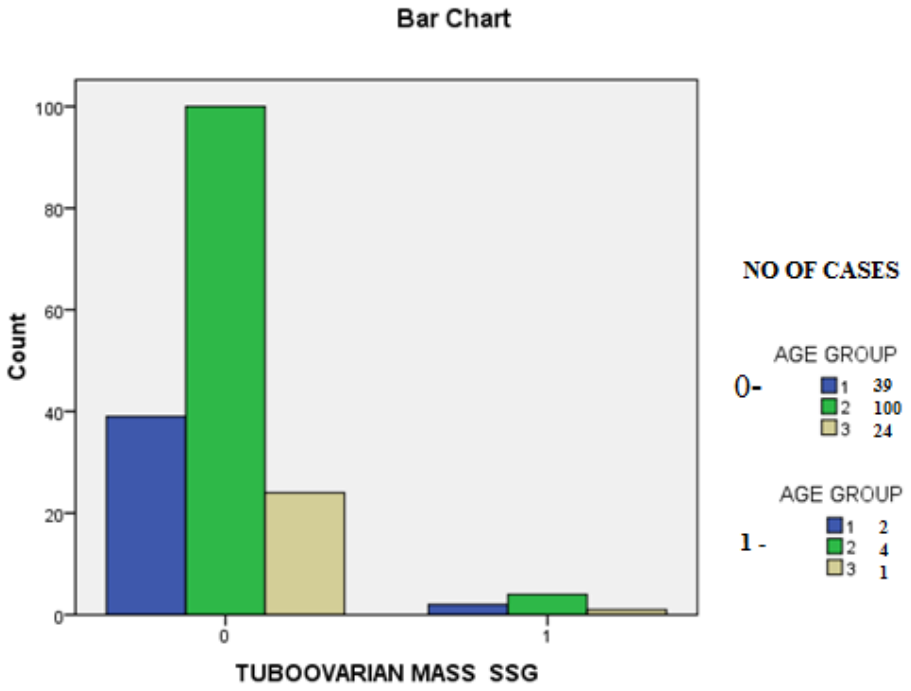
	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.420[a]	2	.298
Likelihood Ratio	2.280	2	.320
Linear-by-Linear Association	2.238	1	.135
N of Valid Cases	170		

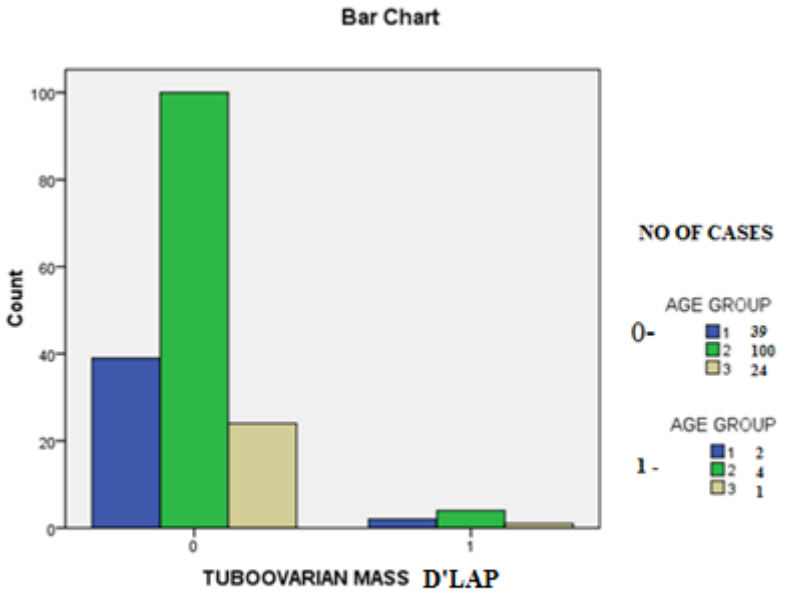


CONCLUSION:

Of the 23 tubal blocks detected by SSG, 5 cases belonged to <25 years,12 cases between 25-30 and 6 cases >30 years. Of the 20 tubal blocks detected by D'LAP, 3 cases were <25 yrs, 12 cases between 25-30 yrs and 5 cases were >30 yrs.

TUBOOVARIAN MASS:

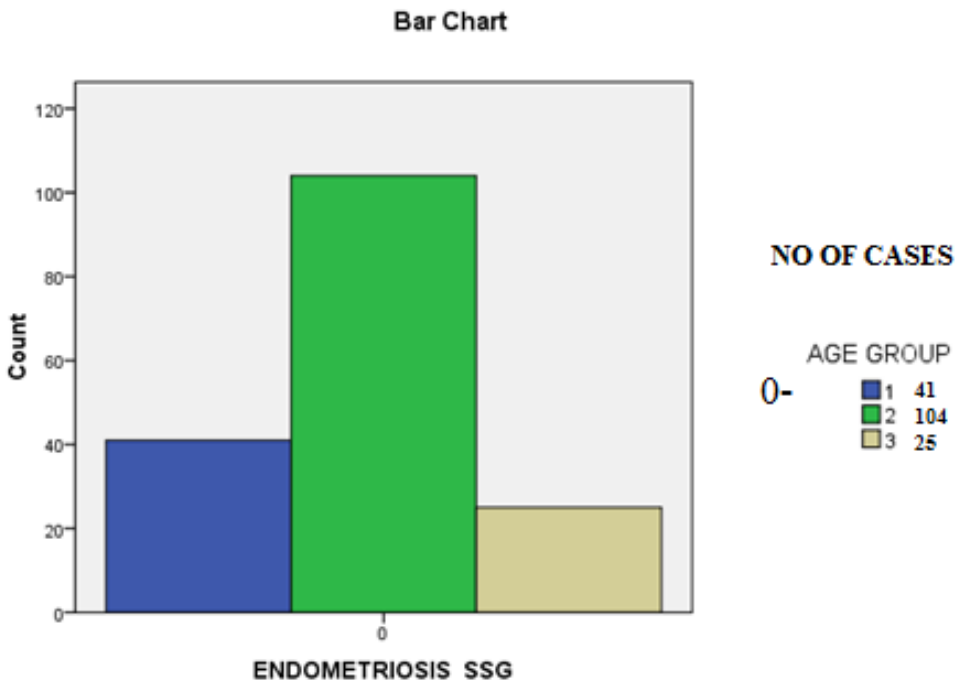


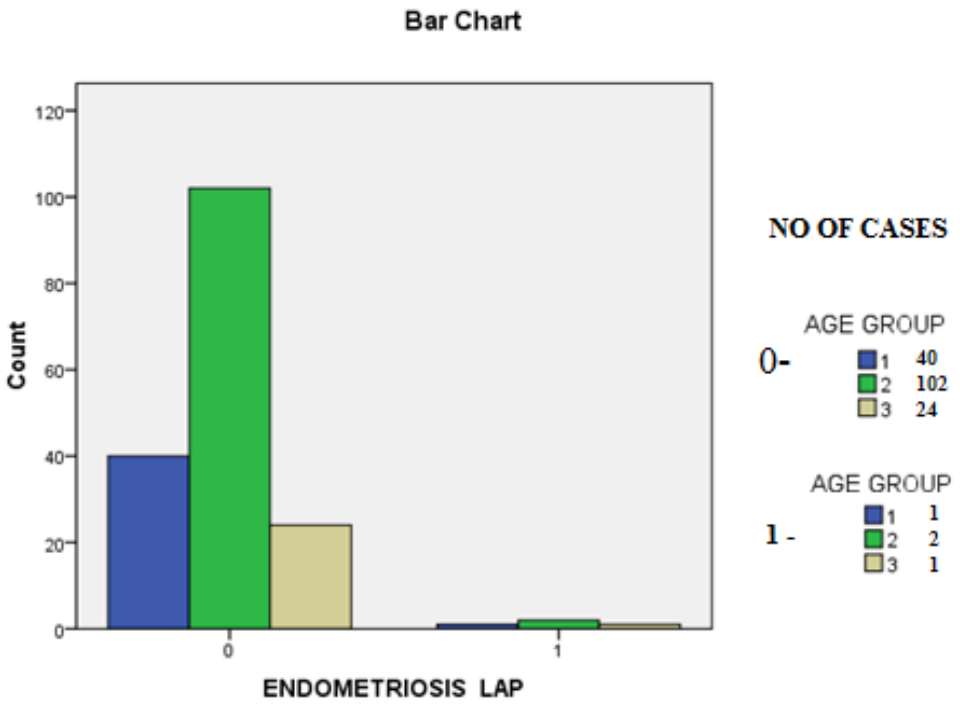


CONCLUSION:

It was found that the detection rate of tuboovarian mass among all the age groups have been similar in both the tests.The incidence among the age groups detected by SSG are 29%,57% and 15% similar to that of LAP.

ENDOMETRIOSIS:

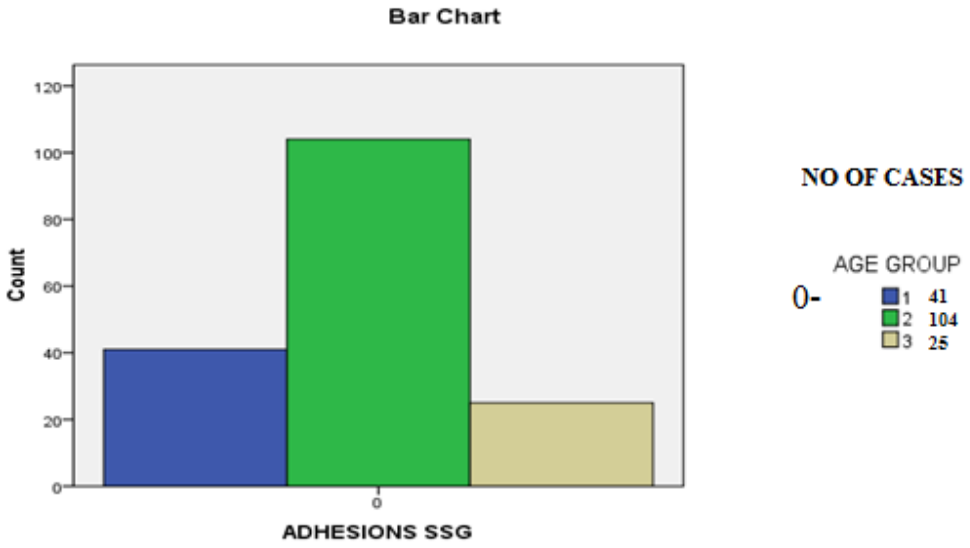


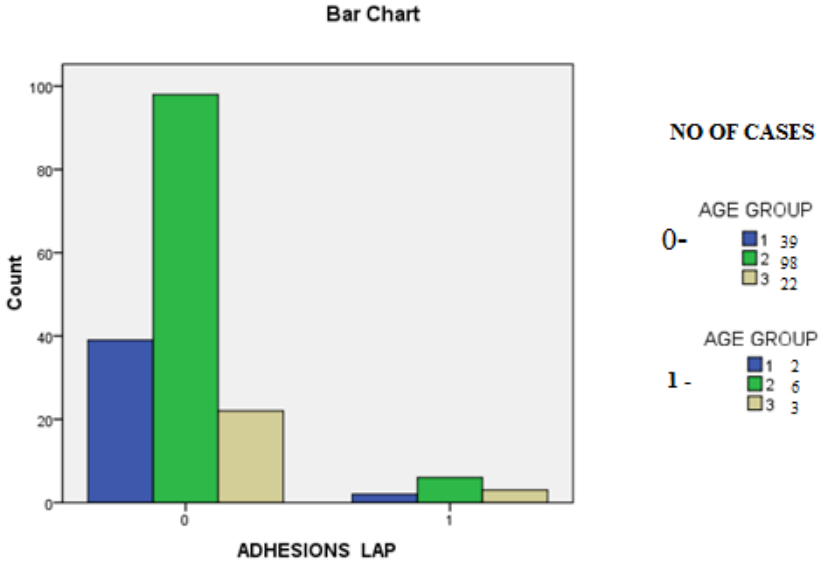


CONCLUSION:

There were 4 cases of endometriosis found in LAP which were not detected in SSG.

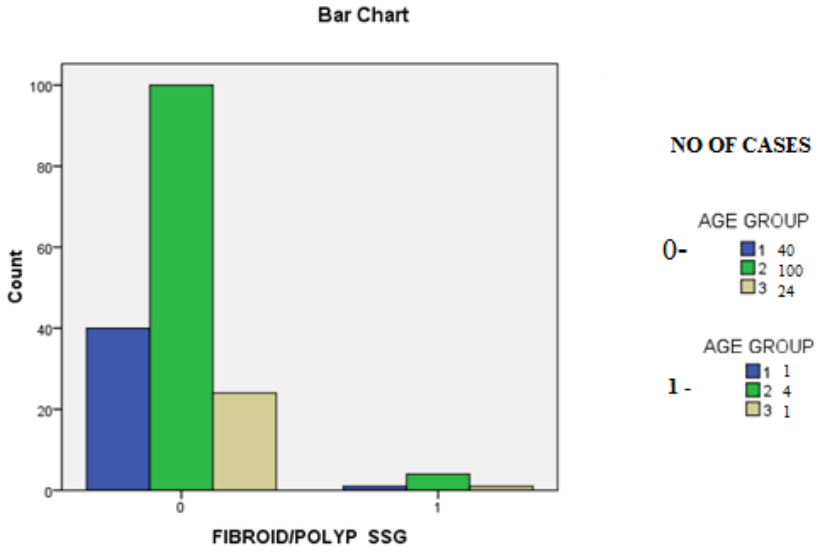
ADHESIONS:

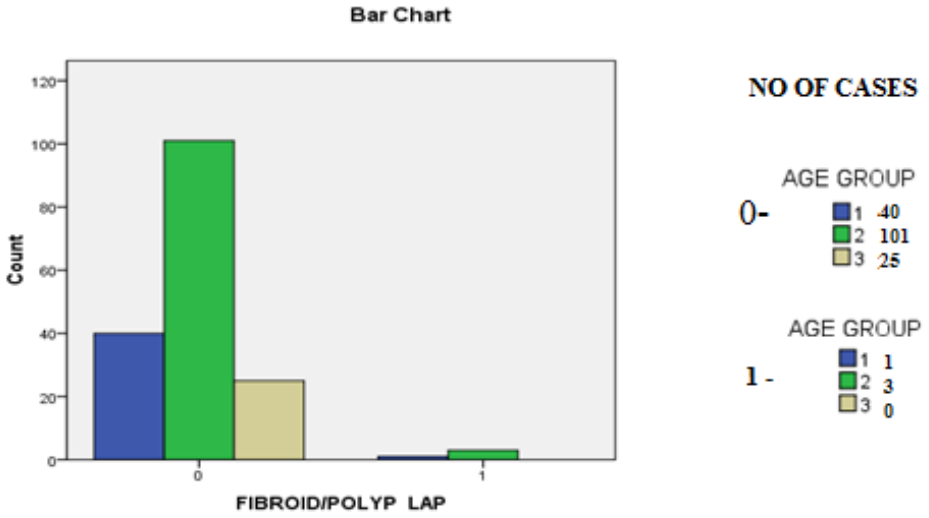

Bar Chart
Count
120
100
80
60
40
20
0
0
ADHESIONS SSG
NO OF CASES
AGE GROUP
0-
1 41
2 104
3 25


Bar Chart
Count
100
80
60
40
20
0
0
1
ADHESIONS LAP
NO OF CASES
AGE GROUP
0-
1 39
2 98
3 22
AGE GROUP
1 -
1 2
2 6
3 3

CONCLUSION : There were 11 cases of adhesions found in LAP which were not detected in SSG.

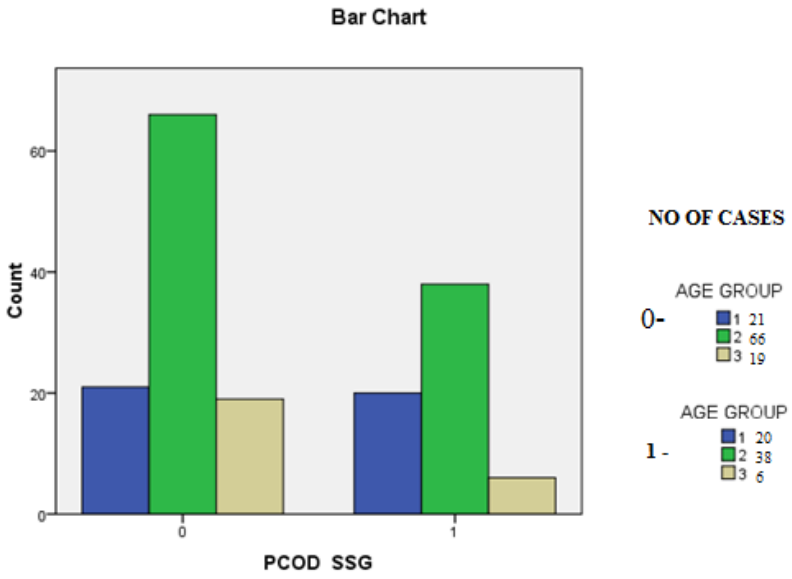
FIBROID:

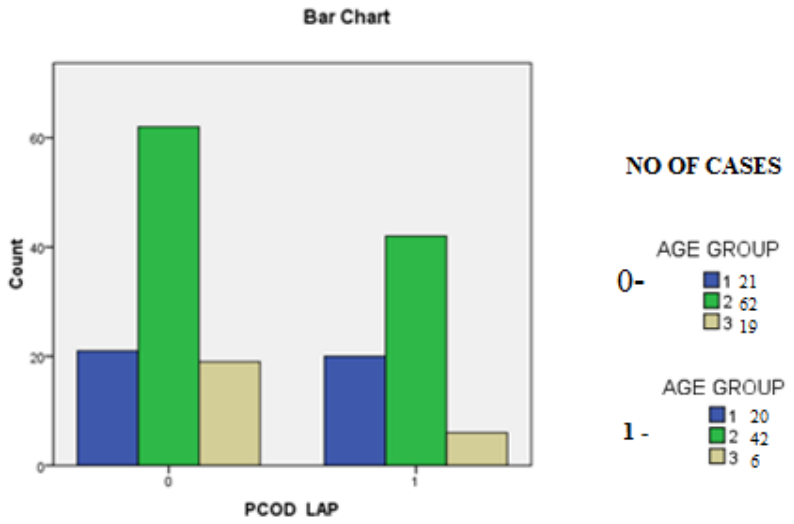




CONCLUSION: The percentage of cases of fibroid in the 3 age groups were 17%, 67%,16% detected by SSG.

PCOD:


Bar Chart
Count
60
40
20
0
0
1
PCOD SSG
NO OF CASES
0-
AGE GROUP
1 21
2 66
3 19
1 -
AGE GROUP
1 20
2 38
3 6



CONCLUSION

The percentage of cases of PCOD detected through SSG in all the 3 age groups were 31%,60% and 9% as compared to that of LAP were 30%,62% and 8%.

SUMMARY

- Taking diagnostic laparoscopy as the gold standard investigation in assessment of tubal patency, our study has compared the sensitivity, specificity, positive and negative predictor values and the diagnostic accuracy of saline infusion sonosalpingography among the patients admitted for subfertility evaluation in KMCH.

- Our study has concluded that since the sensitivity is 96.67%, specificity is 90%,positive predictive value is 98.64 and negative predictive value is 78.26,diagnostic accuracy as estimated by the Wilsons score is 95.88% ,SSG can be used as the initial test for the assessment of tubal patency in subfertile patients.

- Also, among the 170 patients who underwent both procedures, requirement of anaesthesia (164- GA; 6 – Spinal) was 100% for diagnostic laparoscopy and 98% of the patients experienced procedure induced discomfort and post operative pain.

- On the other hand, there was no requirement of anaesthesia for SSG. About 1% complained of procedural discomfort and post procedure pain was reported in <1%.

- There was no case report of pain shock or anaphylaxis associated with this procedure.

- SSG also reported to have an excellent diagnostic accuracy in the detection of endometrial polyp, submucous fibroids, tuboovarian mass, PCOS and other endometrial pathologies.

- However according to our study, SSG failed to diagnose adhesions and endometriosis as compared to D'lap.

DISCUSSION

- **BJOG:** An International Journal of Obstetrics & Gynaecology, Volume 8, Issue 10, pages 1031–1036, October 1991 suggested that sonography as better than HSG as a method of assessing tubal patency as there is no risk of radiation exposure or contrast induced anaphylaxis.

- An article published in ultrasound obstet. Gynecol. 7 (1996) 43-48 suggested the routine use of SSG in tubal patency assessment as it has a similar concordance as that of classical tubal tests of patency.

- J.A.Hamilton et.al in human reproduction volume 13,1998,no. 9 suggests the routine use of SSG in the evaluation of infertility patients.

- R.S. Sankpal et al._International Journal of Gynecology & Obstetrics 73(2001)125_129 stated "The widespread use of sonohysterography in many countries throughout the world suggests that this technique may indeed be a useful primary screening test for tubal patency. This protocol would give preliminary information about uterine anomalies, mapping of

uterine leiomyomas and assessment of the ovaries. Patients with suspect occlusion on contrast SHSG could then undergo laparoscopy with chromopertubation as a secondary test."

- S. Alborzi et al. / International Journal of Gynecology and Obstetrics 82 (2003) 57–62 suggest "the results of this study confirm that SHSG is an easy and reliable method for assessing tubal patency and uterine cavity normalcy without special instrumentation, on an outpatient basis. The procedure is relatively painless and does not require specific medication. At present, it seems that SHSG can replace HSG as a primary method of examination for infertility."

- H.O. Hamed et al. / International Journal of Gynecology and Obstetrics 105 (2009) 215–217 suggests the use of contrast enhanced sonohysterography where there is use of contrast agent in the place of saline,as the first line investigation in tubal patency testing and detecting uterine pathology.

- Our study is similar to that of Masomeh Hajishafiha, MD, Taher Zobairi, MD, Vahide Rasoli Zanjani, MD,Mohammad Ghasemi-

Rad, Zahra Yekta, MD, Nikol Mladkova, MD, who published in J Ultrasound Med 2009; 28:1671–1677 - " On the basis of availability, accessibility, associated risks, and costs, we consider SHG with saline instillation the most efficient first-line diagnostic tool for evaluation of fallopian tube obstruction. It would be advisable to incorporate SHG in routine workups of all couples with female factor infertility, thus reducing the risk of exposure to ionizing radiation and potential development of allergic reactions to iodinated contrast media used during HSG. Also, unlike costly and invasive hysteroscopy, which is associated with a high level of discomfort for the patient, SHG is a simple, cost-effective procedure that is easy to perform, is associated with a low risk of side effects, does not require anesthesia, and causes less discomfort to the patient in comparison with other diagnostic methods."

- Our study is similar to the study by **A.K. P. Ranaweera *et al*., Sch. J. App. Med. Sci., 2013; 1(2):122-130 who stated that "** sensitivity (84.9%) and specificity (81.8%) of the method in diagnosing tubal patency obtained in the present study also support the role of this method as a reliable screening test to assess tubal patency"

CONCLUSION

- At the end of our study, we have concluded that SSG which has an excellent sensitivity, specificity, Positive predictive value, negative predictive value and diagnostic accuracy can be used as the initial test in the assessment of tubal patency among subfertile patients, as there is no risk of anaesthesia or post operative complications as that of D'LAP.

- Also comparison to HSG, there is no radiation exposure or fear of contrast induced anaphylaxis. Hence SSG can be safely used instead of HSG.

- Other associated uterine and ovarian pathologies like endometrial polyps, submucous myoma, PCOD can also be safely yet accurately diagnosed using SSG.

- However, in the presence of sustained subfertility for more than 6 months despite treatment and normal SSG, patients should be posted for diagnostic laparoscopy in view of adhesions or other unknown cause for subfertility.

BIBLIOGRAPHY

1. BJOG: An International Journal of Obstetrics & Gynaecology, Volume 98, Issue 10, pages 1031–1036, October 1991
2. Ultrasound obstet. Gynecol. 7 (1996) 43-48
3. J.A.Hamilton et.al in human reproduction volume 13,1998,no. 9
4. R.S. Sankpal et al._International Journal of Gynecology & Obstetrics 73(2001)125_129
5. S. Alborzi et al. / International Journal of Gynecology and Obstetrics 82 (2003) 57–62
6. H.O. Hamed et al. / International Journal of Gynecology and Obstetrics 105 (2009) 215–217
7. Masomeh Hajishafiha, MD, Taher Zobairi, MD, Vahide Rasoli Zanjani, MD,Mohammad Ghasemi-Rad, Zahra Yekta, MD, Nikol Mladkova, MD, who published in J Ultrasound Med 2009; 28:1671–1677
8. A.K. P. Ranaweera et al., Sch. J. App. Med. Sci., 2013; 1(2):122-130
9. Seal SL, Ghosh D, Saha D. Comaparative evaluation of sonosalpingography, hysterosalpingography and laparoscopy for determination of tubal patency. J Obstet and Gynecol India March/April 2007;57(2):158-61.

10. Suttipichate J et al. Transvaginal saline sonosalpingography for the assessment of tubal patency. VOL. 14, NO. 3, SEPTEMBER 2002
11. A. Dwivedi, M. Jain, S. Tripathi, S. Garg and S. Jain, "Diagnostic Accuracy of Sonosalpingography for Assessing Tubal Pathology in Women with Secondary Infertility Taking Laparoscopy as Gold Standard,"Surgical Science, Vol. 3 No. 8, 2012, pp. 414-417. doi: 10.4236/ss.2012.38082.
12. Seal Subrata Lall, Ghosh Debdatta, Saha Debdas, Bhattacharya Ajit Ranjan,Ghosh Suhash, Mitra SupratimJ Obstet Gynecol India Vol. 57, No. 2 : March/April 2007.
13. Novaks textbook of gynaecology – 14th edition
14. Clinical Gynecologic Endocrinology and Infertility 6th ed: Leon Speroff, Robert H. Glass, Nathan G. Kase, 1999 Lippincott Williams & Wilkins
15. Williams textbook of gynaecology
16. Hammond MG, Talbert LM, eds. Infertility: practical guide for the physician,
17. 2nd ed. Oradell, NJ: Medical Economics, 1985
18. Moghissi K, ed. Current concepts in infertility. Clin Obstet Gynecol 1989;22: 9-1 89

19. Yoder IC. Hysterosalpingography and pelvic ultrasound: imaging in infertility and gynecology. Boston: Little, Brown, 1988

20. Clark RL, Keefe B. Infertility: imaging of the female. Urol Radiol 1989;1 1:233-237

21. Winfield AC, Wentz AC. Diagnostic imaging of infertility. Baltimore: Williams & Wilkins, 1987

22. Hamilton CJCM, Evers JLH, de Haan J. (Jtrasound increases the prognostic value of the postcoital test. Gynecol Obstet Invest 1986;21 : 80-88

23. Daly DC, Aeuter K, Cohen 5, Mastroianni J. Follicle size by ultrasound versus cervical mucus quality: normal and abnormal patterns in spontaneous cycles. Fertil Steril 1989;51 :598-603

24. Check JH, Chase JS, Wu C-H, Goldberg BB, Adelson HG, Rankin A.

25. AJR:159, August 1992 imaging evaluation of infertility in women 261

26. Diagnosis and treatment of the cervical factor. II. Employment of pelvic sonography in diagnosis. mt J Fertli 1986;31 :362-364

27. Mendelson EB, Bohm-Velez M, Joseph N, Neiman HL. Gynecologic imaging:Comparison of transabdominal and transvaginal sonography. Radiology 1988;166:321-324

28. Dudiak CM, Turner DA, Patel 5K, Archie JT, Silver B, Norusis M. Uterine leiomyomas in the infertile patient: preoperative localization with MA imaging versus US and hysterosalpingography. Radiology 1988;167:627-630

29.. Mintz MC, Grumbach K. Imaging of congenital uterine anomalies. Semin ultrasound CTMR 1988;9:167-174

30. Reuter KL, Daly DC, Cohen SM. Septate versus bicomuate uteri: errors in imaging diagnosis. Radiology 1989;172:749-752

31. McCarthy S. Magnetic resonance imaging in the evaluation of infertile women. Magn Reson Q 1990;6:239-249

32. Camngton BM, Hricak H, Nuruddin AN, Secaf E, Laros AK, Hill EC. Mullerian duct anomalies: MR imaging evaluation. Radiology 1990;176:71 5-720

33. Mintz MC, Thickman Dl, Gussman 0, Kressel HY. MR evaluation of uterine anomalies. AJR 1987;148:287-290

34. Kumpe DA, Zwerdlinger SC, Aothbarth II, Durham JD, Albrecht BH.

35. Proximal fallopian tube occlusion: diagnosis and treatment with transcervical fallopian tube catheterization. Radiology 1990;177: 183-1 87

36. Thurmond AS. Selective salpingography and fallopian tube recanalization.AJR 1991;156:33-38

37. Blumenfeld Z, Yoffe N, Bronshtein M. Transvaginal sonography in infertility and assisted reproduction. Obstet GynecolSurv 1990;46:36-49

38. Rasmussen F, Larsen C, Justesen P. Fallopian tube patency demonstrated at ultrasonography. Acta Radiol 1986;27:61-63

39. Angtuaco TL, Boyd CM, London SN, et al. Technetium-99m hysterosalpingography in infertility: an accurate alternative to contrast hysterosalpingography, radioGraphics 1989;9: 115-128

40. Deichert U, Schlief A, van de Sandt M, Juhnke I. Transvaginal hysterosalpingo-contrast-sonography (Hy-Co-Sy) compared with conventional tubal diagnostics. Hum Reprod 1989;4:418-424

41. Schlief A, Deichert U. Hysterosalpingo-contrast sonography of the uterus and fallopian tubes: results of a clinical trial of a new contrast medium in 120 patients. Radiology 1991;178:213-215

42. Peters AJ, Coulam CB. Hysterosalpingography with color Doppler ultrasonography.Am J Obstet Gynecol 1990;1 64:1530-1534

43. Karasick 5, Goldfarb AF. Peritubal adhesions in infertile women: diagnosiswith hysterosalpingography. AJR 1989;1 52:777-779

44. Itskovitz J, Boldes A, Levron J, Thaler I. Transvaginal ultrasonography in the diagnosis and treatment of infertility. JCU 1990;18:248-256

45. Hackeloer BJ. Follicular size assessment by ultrasound. In: Sanders AC,

46. James AE Jr, eds. The principles and practice of ultrasonography in obstetrics and gynecology, 3rd ed. Norwalk, CT: Appleton-Century-Crofts,1985:517-596

47. Daly DC. Treatment validation of ultrasound-defined abnormal follicular dynamics as a cause of infertility. Fertil Steril 1989;51 :51-57

48. Bonilla-Musoles F, Pardo G, Perez-Gil M, Serra V, Pellicer A. Abdominal ultrasonography versus transvaginal scanning: accuracy in follicular development,evaluation and prediction for oocyte retrieval in stimulated cycles. 1989;17:469-473

49. Eissa MK, Sawers AS, Docker MF, Lynch 55, Newton JR. Characteristics and incidence of dysfunctional ovulation pattems detected by ultrasound.Fertil Steril 1987;47:603-612

50. Hackeloer BJ. The role of ultrasound in female infertility management.Ultrasound Med Biol 1984;1 0:35-50

51. Mendelson EB, Friedman H, Neiman HL, Calenoff L, Vogelzang AL, Cohen MA. The role of imaging in infertility management. AJR 1985;1 44:415-420

52. Zandt-Stastny 0, Thorsen MK, Middleton WD, et al. Inability of sonography to detect imminent ovulation. AJR 1989;1 52:91-95

53. Fleischer AC, Herbert CM, Hill GA, Kepple DM. Transvaginal sonography: applications in infertility. Semin Ultrasound CT MR 1990;1 1:71-81

54. Forrest TS, Elyaderani MK, Muilenburg Ml, Bewtra C, Kable WT, Sullivan P. Cyclic endometrial changes: US assessment with histologic correlation.Radiology 1988;1 67:233-237

55. Fleischer AC, Mendelson EB, Bohm-Velez M, Entman SS. Transvaginal and transabdominal sonography of the endometrium. Semin Ultrasound CTMR 1988;9:81-101

56. Yoshimitsu K, Nakamura G, Nakano H. Dating sonographic endometrial images in the normal ovulatory cycle. Int J Gynecol Obstet 1989;28:33-39

57. Brandt TO, Levy EB, Grant TH, Marut E, Leland J. Endornetrial echo and its significance in female infertility. Radiology 1985;157:225-229

Evers JLH; Female subfertility. Lancet, 2002; 360: A.K. P. Ranaweera et al., Sch. J. App. Med. Sci., 2013; 1(2):122-130

58. Bhattacharya S; Infertility. In: Edmonds K, editor.

59. Dewhurtst's textbook of Obstetrics & Gynaecology. 7edition, UK: Blackwell Publishing, 2007: 440 – 60.marakoon S; Infertility in the district of Colombo:University of Colombo. 1994.

60. NICE guideline; Fertility: assessment and treatment for people with fertility problems London: National Institute for Clinical Excellence. 2004.

61. Corson SL; The role of laparoscopy in the infertility workup. J. Reprod. Med., 1977; 18(3):127 – 31,Chapron C, Querleu D, Bruhat MA, Madelenat P,

62. Fernandez H and Pierre F; Surgical complications of diagnostic and operative gynaecological laparoscopy: a series of 29,966 cases. Hum. Reprod., 1998; 13: 867 -72.

63. Swart P MB, van Beurden M, van der Veen F, Redekop WK and Bossuyt PM; The accuracy of hysterosalpingography in the diagnosis of tubal pathology : a meta-analysis. Fertil. Steril., 1995; 64:486 - 91.

64. 8. Ayida G KS, Barlow D and Chamberlain P; A comparison of patient tolerance of hysterosalpingocontrast sonography (HyCoSy) with Echovist-200 and X-ray hysterosalpingography for outpatient investigation of infertile women. Ultrasound Obstet.Gynecol., 1996; 7(3): 201 - 4.

65. Tina SRG; Fallopian Tubal Patency Assessed by Ultrasound Following Fluid Injection. Radiology, 1984;152: 507 - 10.

66. Tufekci EC, Girit S, Bayirli E, Durmusoglu F and Yalti S; Evaluation of tubal patency by transvaginal sonosalpingography. Fertil. Steril., 1992; 57(2): 336-40.

67. Holz K BR and Schurmann R; Ultrasound in the assessment of tubal patency: a meta-analysis of three comparative studies of Echovist 200 including 1007 women. Zentralbi. Gyna-kol., 1997; 1(19): 366 - 73.

68. 12. Tanawattanacharoen SSS, Uerpairojkit B,Wisawasukmongchol W, Boonkasemsanti W and Virutamasen P; Transvaginal HyCoSy compared with chromolaparoscopy: a preliminary report. J. Med.Assoc. Thailand, 1998; 81: 520 - 6.

69. Kalogirou DAG, Botsis D, Kassanos D, Vitoratos N and Zioris C; Is colour doppler necessary in the evaluation of tubal patency by HyCoSy?. Clin. Exp.Obstet. Gynecol., 1997; 24: 101 - 3.

70. http://www.radiologyinfo.org.

71. Muller HJ; Damage to posterity by irradiation of the gonads. Am. J. Obstet. Gynecol., 1954; 67: 467 - 83.

72. http://www.protecheyewear.com

73. Phil Lieberman RLS and George Treadwell;Radiocontrast reactions. Clinical Reviews in Allergy and Immunology, 1986; 4(2): 229 - 45.

74. Chenia F HGJ, Moolla S and Oratis P; Sonographic hydrotubation using agiated saline:a new technique for improving fallopian tube visualization. The British Journal of Radiology, 1997; 70: 833 - 6.

75. Gramiak PMS R and Kramer DH; Ultrasound cardiography: contrast studies in anatomy and function.Radiology, 1969; 92: 939 - 48.

76. Stewart MJ; Contrast Echocardiography. Heart, 2003;89(3): 342 - 8.

77. Patil M; Assessing tubal damage. J. Hum. Reprod. Sci.,2009; 2:2 - 11.

78. AUMenken J, Trussell J, Larsen USO; Age and infertility. Science, 1986; 233(4771): 1389.

79. Volpi E, De Grandis T, Rustichelli S, Zuccaro G, patriarca A and Sismondi P; A new technique to test tubal patency under transvaginal sonographic control.Acta Obstet. Gynecol. Scand., 1994; 73(10): 797-801.

80. Heikkinen H, Tekay A, Volpi E, Martikainen H and Jouppila P; Transvaginal salpingosonography for the assessment of tubal patency in infertile women: methodological and clinical experiences. Fertil. Steril.,1995; 64(2): 293-8.

81. Spalding H, Tekay A, Martikainen H and Jouppila P;Assessment of tubal patency with transvaginal salpingosonography after treatment for tubal pregnancy.Hum. Reprod., 1997; 12(2): 306-9.

82. Inki PPP, Anttila L. Vaginal sonosalpingography in the evaluation of tubal patency. Acta Obstet. Gynecol.Scand., 1998; 77(10): 978 - 82.

83. Ahinko-Hakamaa K, Huhtala H and Tinkanen H; The validity of air and saline hysterosalpingo-contrast sonography in tubal patency investigation before insemination treatment. Eur. J. Obstet. Gynecol.Reprod. Biol., 2007; 132(1): 83-7.

84. Evers JL, Land JA and Mol BW; Evidence-based medicine for diagnostic questions. Semin. Reprod. Med., 2003; 21(1): 9-15.

85. Halkin A, Reichman J, Schwaber M, Paltiel O and Brezis M; Likelihood ratios: getting diagnostic testing into perspective. QJM, 1998; 91(4): 247-58.

86. Goldberg TF and M Attaran; Sonohysterographic evaluation of uterine abnormalities noted on hysterosalpingography. Human Reproduction; 1997;12(10): 2151 - 3.

87. Brown SECC, Schnorr J, Toner JP, Gibbons W and Oehninger S; Evaluation of outpatient hysteroscopy,saline infusion hysterosonography and hysterosalpingography in infertile women: a prospective, randomized study. Fertil. Steril., 2000;74(5): 1029 - 34.

88. Lachlan deCrespigny RK and Dorothy McGinnes;Saline Infusion Sonohysterosalpingography, an Underutilized Technique. Australian

and NewZealand journal of Obstetrics and Gynaecology, 2008; 37(2):206 - 9.

89. Hovav Y, Hornstein E, Almagor M and Yaffe C;Diagnostic laparoscopy in primary and secondary infertility. J. Assist. Reprod. Genet., 1998; 15(9): 535-7.

90. Royal College of Obstetricians and Gynaecologists (RCOG); The investigation and management of endometriosis. London(UK): Royal College of Obstetricians and Gynaecologists (RCOG), 2006.

91. Cepni I, Ocal P, Erkan S, Saricali FS, Akbas H, Demirkiran F; Comparison of transvaginal sonography,saline infusion sonography and hysteroscopy in the evaluation of uterine cavity pathologies.

92. A.K. P. Ranaweera et al., Sch. J. App. Med. Sci., 2013; 1(2):122-130

93. NewZealand journal of Obstetrics and Gynaecology,2005; 45(1): 30-5.

94. Dzotsenidze TN, Davarashvili DI, Nikolaishvili TG,Peradze DG, Datunashvili ED; Contrast sonohysterosalpingography in the study of endometrial abnormalities and tubal patency in infertile patients.Georgian Med. News., 2006; 139:61-3.

95. Hamed HO, Shahin AY and Elsamman AM; Hysterosalpingo-contrast sonography versus radiographic hysterosalpingography in the

evaluation of tubal patency. Int. J. Gynaecol. Obstet., 2009; 105(3):215-7.

96. Friberg B and Joergensen C; Tubal patency studied by ultrasonography. A pilot study. Acta Obstet. Gynecol.Scand., 1994; 73(1): 53-5.

97. Thomas K and Simms I; Chlamydia trachomatis in subfertile women undergoing uterine instrumentation.How we can help in the avoidance of iatrogenic pelvic inflammatory disease?, Hum. Reprod., 2002; 17: 1431 -2.

PROFORMA

NAME:

AGE:

ADDRESS:

EDUCATION:

OCCUPATION:

PARITY:

OP/IP NO

DATE OF VISIT:

CHIEF COMPLAINTS:

DURATION OF PRESENTING ILLNESS:

HISTORY OF PRESENTING ILLNESS:

MENSTRUAL HISTORY:

MARITAL HISTORY:

COITAL HISTORY:

PAST HISTORY:

TREATMENT HISTORY:

OBSTETRICS HISTORY:

FAMILY HISTORY:

EXAMINATION:

HT : WT : BMI:

PALLOR :

BREAST:

THYROID :

VITALS :

PR:

T:

BP:

P/A :

P/S :

P/V:

PROCEDURE: DURATION OF PROCEDURE:

1. Sonosalpingography:
2. Diagnostic laparoscopy:

FINDINGS:

Pathology	Sonosalpingography	laparoscopy
Tubal patency - Rt		
Tubal patency - Lt		
Tuboovarian mass		
Endometriosis		
Peritubal adhesions		
Fibroid uterus		
Ovarian cyst/PCOD		

	Sonosalpingography	Laparoscopy
Requirement of Anaesthesia		
Procedure Discomfort		
Post Procedure Pain		

SIGNATURE OF THE INVESTIGATOR:

SIGNATURE OF THE GUIDE:

INVESTIGATIONS:

- CBC,RFT,URINE R/E
- HIV, VDRL, HbSAg
- BLOOD G/T
- ECG, CHEST XRAY, MANTOUX
- THYROID PROFILE
- SEMEN ANALYSIS
- USG ABD and PELVIS

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு : A COMPARATIVE ASSESSMENT OF SONOSALPINGOGRAPHY (SSG) AND DIAGNOSTIC LAPAROSCOPY FOR DETERMINATION OF TUBAL PATENCY IN CASES OF PRIMARY AND SECONDARY SUBFERTILITY.

Department of Obstetrics and Gynaecology, KMCH

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் வயது : **பங்கு பெறுபவரின் எண் :**

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கிறேன்.எந்த காரணத்தினாலோ எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளல்லாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்கமாட்டேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம்

இடம் :

தேதி :

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் :

சாட்சியாளரின் கையொப்பம்

இடம் :

தேதி :

ஆய்வாளரின் கையொப்பம் :

இடம் : தேதி :

MASTER CHART

S.NO	PATIENT NAME	AGE	TYPE	TUBAL PATENCY		TUBOOVARIAN MASS		ENDOMETRIOSIS		ADHESIONS		FIBROID/POLYP		PCOD	
				SSG	LAP	SSG	LAP	SSG	LAP	SSG	LAP	SSG	LAP	SSG	LAP
1	BHUVANA	21	I	1	1	0	0	0	0	0	0	0	0	1	1
2	VASANTHA	24	I	1	1	0	0	0	0	0	0	0	0	0	0
3	ASHA	23	I	1	1	1	1	0	0	0	0	0	0	0	0
4	MADHAVI	23	I	0	1	0	0	0	0	0	1	0	0	1	1
5	VALLI	21	I	1	1	0	0	0	0	0	0	0	0	1	1
6	WAHEEDA	30	II	1	1	0	0	0	0	0	0	0	0	0	1
7	SAVITHRI	29	I	1	1	0	0	0	0	0	0	0	0	1	1
8	BUVANAMMA	27	I	0	0	0	0	0	0	0	0	0	0	1	1
9	KARPAGAM	22	I	1	1	0	0	0	1	0	0	0	0	1	1
10	BACKIYAM	29	I	1	1	0	0	0	0	0	0	1	1	0	1
11	MARIYAMMAL	21	II	1	1	0	0	0	0	0	0	0	0	1	1
12	MAHALAKSHMI	24	I	0	1	0	0	0	0	0	0	0	0	1	1
13	KAMALA	25	I	1	1	0	0	0	0	0	0	0	0	1	1
14	SUBAMMA	34	I	1	1	0	0	0	0	0	0	0	0	0	0
15	SARASWATHY	25	I	1	1	0	0	0	0	0	0	0	0	0	0
16	RANI	31	II	0	0	1	1	0	0	0	1	0	0	0	0
17	GOKILA	29	I	1	1	0	0	0	0	0	0	1	1	0	0
18	BOOMA	27	I	1	1	0	0	0	0	0	0	0	0	1	1
19	PRIYA	26	I	0	1	0	0	0	0	0	0	0	0	1	1
20	SABURNISHA	23	II	1	1	0	0	0	0	0	0	0	0	0	0
21	DEVIBALA	25	I	1	1	0	0	0	0	0	0	0	0	0	0
22	FARITHABANU	27	I	1	1	0	0	0	0	0	0	0	0	0	0
23	BACKIYA	28	I	1	1	0	0	0	0	0	0	0	0	0	1
24	POONGAVANAM	27	I	1	1	0	0	0	0	0	0	0	0	0	0
25	JOTHI	31	I	1	1	0	0	0	0	0	0	0	0	0	0
26	MAHADEVI	31	I	0	0	0	0	0	0	0	0	0	0	0	0
27	MARY	32	I	0	0	0	0	0	0	0	0	0	0	1	1
28	LEELAVATHI	29	II	0	0	0	0	0	0	0	0	0	0	1	1
29	ARASI	26	I	0	0	0	0	0	0	0	1	0	0	1	1
30	VIJAYALAKSHMI	23	II	0	0	0	0	0	0	0	0	0	0	0	0
31	AMUDHA	28	I	1	1	0	0	0	0	0	0	0	0	0	0
32	GOMATHI	29	II	1	1	0	0	0	0	0	0	0	0	0	0
33	SAROJA	28	I	1	1	0	0	0	0	0	0	0	0	1	1

34	CHOKKI	36	II	1	1	0	0	0	0	0	0	0	0	0	0
35	VENI	33	II	1	1	0	0	0	0	0	1	0	0	0	0
36	MABOOBEE	25	I	1	1	0	0	0	0	0	0	0	0	0	0
37	AESHA	22	I	1	1	1	1	0	0	0	0	0	0	0	0
38	PADMINI	26	I	1	1	0	0	0	0	0	0	0	0	1	1
39	KALAISELVI	27	I	1	1	0	0	0	0	0	0	0	0	0	0
40	AKHILA	27	I	1	1	0	0	0	0	0	0	1	0	0	0
41	SUGUMARI	29	II	1	1	0	0	0	0	0	0	0	0	1	1
42	KANIMOZHI	28	II	1	1	0	0	0	0	0	0	0	0	1	1
43	RUPAVATHY	31	II	1	1	0	0	0	0	0	1	0	0	0	0
44	LALITHA	32	II	1	1	0	0	0	0	0	0	0	0	0	0
45	ARPUTHAM	27	I	1	1	0	0	0	0	0	0	0	0	0	0
46	KURUVAMMA	31	I	0	0	0	0	0	0	0	0	0	0	0	0
47	BHAVANI	21	I	0	0	0	0	0	0	0	0	0	0	1	1
48	SEETHA	25	I	1	1	0	0	0	0	0	0	0	0	0	0
49	KATHAYI	27	I	1	1	0	0	0	0	0	0	0	0	0	0
50	ANANDHI	26	I	1	1	0	0	0	0	0	0	0	0	1	1
51	KUNAVATHI	29	II	0	0	0	0	0	0	0	0	0	0	0	0
52	NILA	30	II	1	1	0	0	0	0	0	0	0	0	0	0
53	MALARKODI	29	I	1	1	0	0	0	0	0	0	0	0	1	1
54	SENGAMALAM	26	I	1	1	1	1	0	0	0	0	0	0	1	1
55	SEMBA	27	I	1	1	0	0	0	0	0	0	0	0	0	0
56	VANI	26	I	1	1	0	0	0	0	0	1	0	0	0	0
57	NAGAVALLI	29	I	1	1	0	0	0	0	0	0	0	0	1	1
58	UMAYUNNISHA	31	II	1	1	0	0	0	0	0	0	0	0	0	0
59	LALITHA	28	II	1	1	0	0	0	0	0	0	0	0	0	0
60	THILAGAVATHY	25	II	1	1	0	0	0	0	0	0	0	0	1	1
61	KUMARI	27	I	1	1	0	0	0	0	0	0	0	0	0	0
62	SANTHA	29	I	1	1	0	0	0	0	0	0	0	0	1	1
63	SUDHA	24	I	1	1	0	0	0	0	0	0	0	0	0	0
64	SUMATHY	23	I	1	1	0	0	0	0	0	0	0	0	0	0
65	GEETHA	23	I	1	1	0	0	0	0	0	0	1	1	1	1
66	MALA	25	I	1	1	0	0	0	0	0	0	0	0	0	0
67	PAVALAM	29	II	1	1	0	0	0	0	0	0	0	0	0	0
68	CHELLAMMA	32	I	0	1	0	0	0	0	0	0	0	0	1	1
69	USHA	25	I	1	1	0	0	0	0	0	0	0	0	1	1
70	SIPPI	26	I	1	1	0	0	0	0	0	0	0	0	1	1
71	PREMA	27	I	1	1	1	1	0	0	0	0	0	0	0	0
72	DEEPA	29	I	1	1	0	0	0	0	0	0	0	0	0	0
73	FATHIMA	27	II	1	1	0	0	0	0	0	0	0	0	0	0

74	PARAMESWARI	31	II	1	1	0	0	0	0	0	0	0	0	0	0
75	LAKSHMI	30	I	1	1	0	0	0	0	0	0	0	0	0	0
76	SENGAMALAM	30	I	1	1	0	0	0	0	0	0	0	0	0	0
77	RADHA	23	I	1	1	0	0	0	0	0	0	0	0	1	1
78	DEEPIKA	29	I	1	1	0	0	0	0	0	0	0	0	1	1
79	PARGAVI	23	I	1	1	0	0	0	0	0	0	0	0	1	1
80	MANORANJITHAM	25	I	1	1	0	0	0	0	0	0	0	0	1	1
81	NAVAMANI	29	I	1	1	0	0	0	0	0	1	0	0	0	0
82	VARALAKSHMI	32	I	1	1	0	0	0	0	0	0	0	0	0	0
83	ANJALI	25	II	1	1	0	0	0	0	0	0	0	0	0	0
84	SUBBULAKSHMI	26	II	1	1	0	0	0	0	0	0	0	0	0	0
85	GANGA	27	II	1	1	0	0	0	0	0	0	0	0	0	0
86	SAMPOORNAM	29	I	1	1	0	0	0	0	0	0	0	0	1	1
87	SARALA	23	I	1	1	0	0	0	0	0	0	0	0	1	1
88	ARPUTHARANI	28	I	1	1	0	0	0	0	0	0	0	0	1	1
89	MEENA	29	I	1	1	0	0	0	0	0	0	0	0	0	0
90	GOWRI	28	I	1	1	0	0	0	0	0	0	0	0	0	0
91	JAMEELA	30	I	1	1	1	1	0	0	0	0	0	0	0	0
92	SAROJA	34	I	1	1	0	0	0	0	0	0	0	0	0	0
93	CHITRA	29	I	1	1	0	0	0	0	0	0	0	0	1	1
94	PRIYA	30	I	1	1	0	0	0	0	0	0	0	0	0	0
95	MYTHILI	28	I	1	1	0	0	0	0	0	0	0	0	0	0
96	SHEELA	31	I	1	1	0	0	0	0	0	0	0	0	0	0
97	SRIDEVI	30	I	1	1	0	0	0	0	0	0	0	0	1	1
98	THAVAMANI	29	I	1	1	0	0	0	0	0	0	0	0	1	1
99	NOORJAHAN	28	I	1	1	0	0	0	0	0	0	0	0	0	0
100	SUSEELA	32	I	1	1	0	0	0	0	0	0	0	0	0	0
101	SULOCHANA	27	I	1	1	0	0	0	0	0	0	0	0	0	0
102	SUBATHRA	29	II	1	1	0	0	0	0	0	0	0	0	0	0
103	MEENAL	27	I	1	1	0	0	0	0	0	0	0	0	1	1
104	BALAMANI	26	II	0	1	0	0	0	0	0	0	0	0	0	0
105	MAYA	29	II	0	0	0	0	0	0	0	0	0	0	0	0
106	MARUTHAYI	28	I	1	1	0	0	0	0	0	1	0	0	0	0
107	SARASWATHY	31	II	1	1	0	0	0	0	0	0	1	0	1	1
108	MALLIGA	29	I	1	1	0	0	0	0	0	0	0	0	0	0
109	VALARMATHI	28	II	1	1	1	1	0	0	0	0	0	0	0	0
110	MALARVIZHI	25	I	1	1	0	0	0	0	0	0	0	0	0	0
111	SELVI	27	I	1	1	0	0	0	0	0	0	0	0	1	1

112	LATHA	26	I	1	1	0	0	0	0	0	0	0	0	0	0
113	KRITHIKA	27	II	0	0	0	0	0	0	0	0	0	0	0	0
114	ESWARI	29	II	0	0	0	0	0	0	0	0	0	0	1	1
115	PRIYANKA	30	II	1	1	0	0	0	0	0	0	0	0	0	0
116	MANJULA	27	I	1	1	0	0	0	0	0	0	0	0	0	0
117	SANDHYA	24	I	1	1	0	0	0	0	0	0	0	0	0	0
118	SHEELA	23	I	1	1	0	0	0	0	0	0	0	0	0	0
119	SUJATHA	25	I	1	1	0	0	0	0	0	0	0	0	1	1
120	KRISHNAVENI	27	I	1	1	0	0	0	0	0	0	0	0	1	1
121	AMALAMARY	31	I	1	1	0	0	0	0	0	0	0	0	0	0
122	MARY	30	I	1	1	0	0	0	0	0	0	0	0	0	0
123	HAMSA	25	II	1	1	0	0	0	0	0	0	0	0	0	0
124	SHANTHI	28	I	1	1	0	0	0	0	0	0	0	0	0	0
125	KASTURI	29	I	1	1	0	0	0	0	0	0	0	0	0	0
126	MEENAMBAL	28	I	1	1	0	0	0	0	0	0	0	0	0	0
127	SHANTHI	25	II	1	1	0	0	0	0	0	0	0	0	1	1
128	ELAVARASI	27	II	1	1	0	0	0	0	0	0	0	0	0	0
129	VASUKI	26	II	1	1	0	0	0	0	0	0	0	0	0	0
130	ANJALIDEVI	27	I	1	1	0	0	0	0	0	0	0	0	0	0
131	NARAYANI	29	I	1	1	0	0	0	0	0	0	0	0	0	0
132	ALAMELU	29	I	1	1	0	0	0	0	0	0	0	0	1	1
133	MEENATCHI	28	I	0	0	0	0	0	0	0	0	0	0	0	0
134	MANGALAM	25	II	1	1	0	0	0	0	0	1	0	0	0	0
135	RAMADEVI	27	II	1	1	0	0	0	0	0	0	0	0	0	0
136	KOLANJI	26	II	1	1	0	0	0	0	0	0	0	0	0	0
137	LAKSMIAMMAL	27	I	1	1	0	0	0	0	0	0	0	0	0	0
138	BHANU	29	I	1	1	0	0	0	1	0	0	0	0	1	1
139	POOVARASI	31	I	1	1	0	0	0	0	0	0	0	0	1	1
140	MARIYAM	33	I	1	1	0	0	0	0	0	0	0	0	1	1
141	ARCHANA	32	I	1	1	0	0	0	0	0	0	0	0	0	0
142	EASWARI	29	I	1	0	0	0	0	0	0	0	0	0	0	0
143	RUKKU	27	I	0	0	0	0	0	0	0	0	0	0	1	1
144	DEVI	20	I	0	0	0	0	0	0	0	0	0	0	1	1
145	SAVITHRI	22	II	1	1	0	0	0	0	0	0	0	0	0	0
146	RAMA	32	I	1	1	0	0	0	0	0	0	0	0	0	0
147	VASUKI	29	I	1	1	0	0	0	0	0	0	0	0	0	0
148	VISALATCHI	27	I	1	1	0	0	0	0	0	0	0	0	0	0
149	NITHYAKALA	25	I	1	1	0	0	0	0	0	0	0	0	0	0
150	RAMANI	27	I	1	1	0	0	0	0	0	0	0	0	1	1
151	MANGALAM	26	I	1	1	0	0	0	0	0	0	0	0	1	1

152	VIJAYA	29	I	1	1	0	0	0	0	0	0	0	0	0	0
153	MOHANA	30	II	1	1	0	0	0	0	0	0	0	0	0	0
154	CHANDRA	31	II	1	1	0	0	0	0	0	0	0	0	1	1
155	KUPPU	29	I	1	0	0	0	0	0	0	0	0	0	0	0
156	HABEEBNISHA	26	I	1	1	0	0	0	0	0	0	0	0	1	1
157	KUMUDHA	30	II	1	1	0	0	0	0	0	1	0	0	1	1
158	CHINNATHAYI	21	I	1	1	0	0	0	0	0	0	0	0	1	1
159	USHARANI	28	I	1	1	0	0	0	0	0	0	0	0	1	1
160	SAGUNTHALA	25	I	1	1	0	0	0	0	0	0	0	0	1	1
161	AANDAL	24	I	1	1	0	0	0	0	0	0	0	0	0	0
162	RASATHI	29	I	1	1	0	0	0	0	0	0	0	0	0	1
163	VADIVU	31	II	0	0	0	0	0	1	0	0	0	0	0	0
164	NAGALAKSHMI	29	I	1	1	0	0	0	0	0	0	1	1	1	1
165	KAMALA	26	I	1	1	0	0	0	0	0	1	0	0	0	0
166	KAMATCHI	30	II	1	1	0	0	0	0	0	0	0	0	0	0
167	RANI	29	I	1	1	0	0	0	0	0	0	0	0	0	0
168	KANAGAVALLI	28	I	0	0	0	0	0	0	0	0	0	0	1	1
169	NAGARATHINAM	28	I	1	1	0	0	0	0	0	0	0	0	1	1
170	TAMILSELVI	26	I	1	1	0	0	0	1	0	0	0	0	1	1
	TOTAL NUMBER OF CASES			**147**	**150**	**7**	**7**	**0**	**4**	**0**	**11**	**6**	**4**	**64**	**68**

KEYS	
0	NO
1	YES

ABBREVIATIONS

SIS	SALINE INFUSION SONOGRAPHY
SSG	SONOSALPINGOGRAPHY
LAP	LAPAROSCOPY
HSG	HYSTEROSALPINGOGRAM
SHG / SHSG	SONOHYSTEROGRAM
PCOS	POLYCYSTIC OVARIAN SYNDROME
PCOD	POLYCYSTIC OVARIAN DISEASE

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.No. 16420/ME-1/Ethics/2013 Dt:06.11.2013
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study on comparative assessment of sonosalpingography (SSG) and diagnostic laparoscopy for determination of tubal patency in cases of primary and secondary subfertility" – For Project Work Submitted by Dr.C.Rajani, MS (O&G), PG Student, KMC, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

GOVT. KILPAUK MEDICAL COLLEGE * Kilpauk, Chennai-10. * 25 NOV 2013

25/11/13.

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,
Chennai

22/11/13